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OF THE

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OF EDINBURGH

VOL. XXX.—NEW SERIES

SESSION 1910-1911

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PREFACE

THE present Volume is the *Thirtieth* of the *New Series*, and contains a record of the work done during the past Session.

That work, as hitherto, embraces the communication of Original Papers; the exhibition of Patients, illustrating rare and interesting forms of disease; and the exhibition of Pathological and other Specimens, so essential to the proper understanding of the morbid changes which take place in the human body.

Nine meetings were held in the year, one of them being a clinical meeting.

CHALMERS WATSON,
Editor.

September 1911.

14156

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	Dr Byrom Bramwell, 23 Drumsheugh Gardens, *President*,	1876
	Dr Edwin Bramwell, 24 Walker Street,	1900
20	Dr N. T. Brewis, 6 Drumsheugh Gardens	1886
	Dr A. J. Brock, 27 Braid Crescent,	1907
	Dr J. J. Graham Brown, 3 Chester Street,	1878
	Dr R. Dods Brown, Royal Asylum, Edinburgh,	1908

		Date of Admission.
	Dr A. H. Buchan, 2 Dean Terrace,	1899
25	Dr Buist, 1 Clifton Terrace,	1877
	Dr T. M. Burn-Murdoch, 14 Charlotte Square,	1886
	Dr J. Burnet, 6 Glengyle Terrace,	1907
	Professor Francis M. Caird, 13 Charlotte Square,	1883
	Dr H. L. Calder, 60 Leith Walk, Leith,	1884
30	Dr James Cameron, 1 Atholl Place,	1895
	Dr A. N. S. Carmichael, 2 Merchiston Avenue,	1902
	Dr Edward Carmichael, 21 Abercromby Place,	1887
	Dr E. W. Scott Carmichael, 40 Palmerston Place,	1901
	Dr George Scott Carmichael, 25 Braid Road,	1903
35	Dr J. Carmichael, 22 Northumberland Street,	1870
	Dr N. Scott Carmichael, 12 Northumberland Street,	1910
	Dr George J. R. Carruthers, 4A Melville Street,	1901
	C. W. Cathcart, Esq., 44 Melville Street,	1883
	Dr J. G. Cattanach, 3 Alvanley Terrace,	1895
40	George L. Chiene, Esq., 23 Alva Street,	1901
	Professor John Chiene, C.B., 21 Alva Street,	1867
	Dr A. E. Chisholm, 7 Claremont Crescent,	1910
	Dr H. M. Church, 36 George Square,	1876
	Dr A. W. Greenhorn Clark, 24 Braid Crescent,	1908
45	Dr R. D. Clarkson, Larbert Institution, Larbert,	1896
	Sir T. S. Clouston, 26 Heriot Row,	1861
	Dr John D. Comrie, 25 Manor Place,	1902
	Dennis Cotterill, Esq., 10 Coates Crescent,	1909
	J. M. Cotterill, Esq., 24 Manor Place, *Vice-President*,	1878
50	Dr John Craig, 71 Bruntsfield Place,	1900
	Dr William Craig, 71 Bruntsfield Place,	1869
	Dr D. M. Crichton, Burgh House, Maryhill, Glasgow,	1906
	Dr David Halliday Croom, 17 Alva Street,	1906
	Professor Sir John Halliday Croom, 25 Charlotte Square,	1870
55	Dr W. J. Crow, 108 Morningside Road,	1908
	Dr G. Matheson Cullen, 50 Minto Street,	1892
	Dr John Cumming, 70 Bruntsfield Place,	1894
	Dr T. B. Darling, 13 Merchiston Place,	1887
	Dr G. A. Davies, 7 West Holmes Gardens, Musselburgh,	1906
60	Dr J. M. Dewar, 24 Lauriston Place,	1885
	Dr Thomas W. Dewar, Kincairn, Dunblane,	1906
	Dr J. T. Dickie, 37 Lauriston Place,	1900
	Dr D. Elliot Dickson, Hill Crescent, Lochgelly, Fife,	1906
	Dr George Dickson, 26 Merchiston Park,	1901
65	Dr W. E. Carnegie Dickson, 92A Findhorn Place,	1902
	Dr Alex. Don, Park House, Nethergate, Dundee,	1909
	Dr C. E. Douglas, Cupar-Fife,	1894
	Dr Kenneth M. Douglas, 19 Chester Street,	1888
	Dr W. B. Dow, Dunfermline,	1879
70	J. W. Dowden, Esq., 48 Manor Place, *Treasurer*,	1893
	Dr W. B. Drummond, 2 Braidburn Terrace,	1900
	Dr James K. Drysdale, 22 Coltbridge Terrace,	1901
	Dr Henry J. Dunbar, 3 Hampton Terrace,	1906
	Dr John A. H. Duncan, 4 Cluny Drive,	1895
75	Dr H. M. Dunlop, 20 Abercromby Place,	1883
	Dr J. C. Dunlop, 33 Chester Street,	1892
	Dr John Eason, 58 Northumberland Street,	1900
	Dr William Elder, 4 John's Place, Leith,	1892
	Dr J. Haig Ferguson, 7 Coates Crescent,	1885
80	Dr Thomas Finlay, 8 Comely Bank Road,	1903
	Dr Andrew F. Fleming, St John's Road, Corstorphine,	1905
	Dr R. A. Fleming, 10 Chester Street,	1890
	Dr A. B. Flett, 60 George Square.	1903
	Dr W. E. Foggie, 4 Airlie Place, Dundee,	1907
85	Dr A. Dingwall Fordyce, 8 Manor Place.	1901

		Date of Admission
	Dr W. Fordyce, 20 Charlotte Square,	1907
	Dr J. S. Fowler, 55 Northumberland Street,	1895
	Dr John Fraser, 13 Heriot Row,	1878
	Dr J. Hossack Fraser, Fernfield, Bridge of Allan,	1895
90	Dr J. S. Fraser, 50 Melville Street,	1905
	Prof. Sir Thomas R. Fraser, 13 Drumsheugh Gardens,	1865
	Dr W. Ernest Frost, 36 Moray Place,	1904
	Dr G. Fyfe, 24 Broughton Place,	1911
	Dr F. Gardiner, 9 George Square,	1900
95	Dr J. H. Gibbs, 12 Coates Crescent,	1905
	Dr G. A. Gibson, 3 Drumsheugh Gardens,	1880
	Dr A. B. Giles, 4 Palmerston Place,	1893
	Dr Alexander Goodall, 7 Mayfield Road,	1901
	Dr D. J. Graham, 26 Rutland Street,	1897
100	Dr F. M. Graham, 16 Mayfield Gardens,	1894
	Dr J. M. Graham, 6 Castle Terrace,	1908
	Dr J. Taylor Grant, 3 Manor Place,	1898
	Dr J. Allan Gray, 107 Ferry Road,	1879
	Dr John L. Green, 23 Minto Street,	1904
105	Professor Greenfield, 7 Heriot Row,	1886
	Dr David M. Greig, 25 Tay Street, Dundee,	1892
	Dr G. L. Gulland, 11 Chester Street,	1888
	Dr James A. Gunn, 18 Morningside Drive,	1906
	Dr A. C. Guthrie, 21 Pilrig Street,	1890
110	Dr William Guy, 11 Wemyss Place,	1890
	Dr William Haldane, Viewforth, Bridge of Allan,	1889
	Dr D. Berry Hart, 5 Randolph Cliff,	1886
	Dr James Harvey, 7 Blenheim Place,	1893
	J. W. B. Hodsdon, Esq., 6 Chester Street,	1883
115	Dr George Hunter, 33 Palmerston Place,	1876
	Dr David Huskie, Moffat,	1899
	Dr R. W. Inkster, 16 Windsor Street,	1895
	Dr Alex. James, 14 Randolph Crescent,	1877
	Dr Allan Jamieson, 35 Charlotte Square, *Vice-President*,	1876
120	Dr Hugh Jamieson, 1 Strathearn Road,	1889
	Dr J. Boyd Jamieson, 43 George Square,	1900
	Dr G. M. Johnston, 7 Wellington Place, Leith,	1888
	Dr R. J. Johnston, 18 George Square,	1897
	Dr R. M'Kenzie Johnston, 2 Drumsheugh Gardens,	1883
125	Dr R. W. Johnstone, 10 Alva Street,	1907
	Dr C. B. Ker, City Hospital, Comiston Road,	1894
	Dr F. K. Kerr, 168 Newhaven Road, Leith,	1910
	Dr George Kerr, 6 St Colme Street,	1885
	Dr J. Lamond Lackie, 7 Randolph Crescent,	1907
130	Dr H. G. Langwell, 4 Hermitage Place, Leith,	1899
	Dr G. Laurence, Annfield House, Dundee,	1910
	Dr David Lawson, Nordrach-on-Dee, Banchory,	1901
	Dr H. A. Leebody, Corstorphine,	1899
	Dr Edwin M. Lithgow, 8 Alva Street,	1901
135	Dr John D. Lithgow, 8 Alva Street,	1901
	Professor Harvey Littlejohn, 11 Rutland Street,	1889
	Emeritus Professor Sir Henry D. Littlejohn, 24 Royal Circus,	1853
	Dr R. Cranston Low, 7 Castle Terrace,	1906
	Dr R. A. Lundie, 55A Grange Road,	1883
140	Dr W. Leslie Lyall, 8 Murrayfield Gardens,	1902
	Dr D. C. A. M'Allum, 11 Greenhill Gardens,	1900
	Dr Angus Macdonald, 27 Manor Place,	1902
	Dr John M'Gibbon, 24 Melville Street,	1899
	Dr C. W. MacGillivray, 15 Charlotte Square, *Vice-President*,	1877
145	Dr A. M. M'Intosh, 30 East Claremont Street,	1904
	Dr George Mackay, 20 Drumsheugh Gardens,	1887
	Dr A. M'Kendrick, 27 Chalmers Street,	1909

		Date of Admission.
	Dr W. Leslie Mackenzie, 1 Stirling Road, Trinity,	1895
	Dr Malcolm M'Larty, 7 Bellevue Place,	1900
150	Dr John Macmillan, 48 George Square,	1897
	Dr C. M'Neil, 4 Torphichen Street,	1909
	Dr John Macpherson, 15 Rutland Square,	1891
	Dr John MacRae, Lynwood, Murrayfield Road,	1893
	Dr Douglas M'Rae, Glengall, Ayr,	1904
155	Dr Alexander M. Malcolmson, St John's Road, Corstorphine,	1905
	Major D. G. Marshall, I.M.S., 25 Buckingham Terrace,	1905
	Dr A. A. Matheson, 41 George Square,	1901
	Dr Roderick M. Matheson, 33 Buccleuch Place,	1902
	Dr Edwin Matthew, 9 Walker Street,	1900
160	Dr J. Halley Meikle, 12 Midmar Gardens,	1906
	Dr Kenmure Melville, 2 Nile Grove,	1908
	Dr D. Menzies, 20 Rutland Square,	1878
	A. Miles, Esq., 20 Walker Street, *Secretary*,	1892
	Dr J. Mill, 188 Ferry Road,	1886
165	A. G. Miller, Esq., 8 Garscube Terrace,	1867
	Dr James Miller, 24 Queen Street,	1910
	Dr W. H. Miller, 51 Northumberland Street,	1895
	Dr W. Morrison Milne, 10 Newington Road,	1905
	Dr John Mowat, 5 Hope Park Terrace,	1885
170	Dr T. Goodall Nasmyth, 27 Palmerston Place,	1884
	Dr H. Oliphant Nicholson, 20 Manor Place,	1900
	Dr John Orr, 6 Strathearn Road,	1895
	Dr W. Basil Orr, 13 Braid Road,	1893
	Dr G. Keppie Paterson, 19 Albany Street,	1889
175	Dr J. V. Paterson, 11 Melville Street,	1900
	Dr Simla Paterson, 147 Bruntsfield Place,	1904
	Dr William Paterson, 8 Hermitage Place, Leith,	1909
	Dr P. Pattison, 4 Novar Crescent, Kirkcaldy,	1907
	Dr C. Balfour Paul, 17 Walker Street,	1904
180	Dr C. M. Pearson, 14 Manor Place,	1902
	Dr H. A. Peddie, 24 Palmerston Place,	1893
	Dr R. W. Philip, 45 Charlotte Square,	1883
	Dr J. H. Harvie Pirie, 5 Castle Terrace,	1905
	Dr Playfair, 5 Melville Crescent,	1874
185	Dr Frederick Porter, 65 Morningside Road,	1904
	Dr W. G. Porter, 16 Manor Place,	1907
	Dr Edmond Price, 1 Middleby Street,	1909
	Dr T. Proudfoot, 30 Lauriston Place,	1889
	Dr Harry Rainy, 16 Great Stuart Street,	1896
190	Dr Douglas G. Reid, Finkenberg, St Barnabas Road, Cambridge,	1905
	Dr H. S. Reid, 6 Kew Terrace,	1910
	Dr James Ritchie, 22 Charlotte Square,	1873
	Dr James Ritchie, 10 Succoth Gardens,	1907
	Dr L. C. Peel Ritchie, 7 Alva Street,	1904
195	Dr W. T. Ritchie, 9 Atholl Place,	1900
	Dr George M. Robertson, Tipperlinn House, Morningside Place,	1890
	Dr Robert Robertson, 26 Royal Circus,	1901
	Dr William Robertson, 16 Stirling Road,	1902
	Dr W. Ford Robertson, 48 Northumberland Street,	1893
200	Dr W. G. Aitchison Robertson, 2 Mayfield Gardens,	1892
	Dr R. M. Ronaldson, 17 Morningside Road,	1895
	Dr T. R. Ronaldson, 8 Charlotte Square,	1877
	Dr A. Aitken Ross, 8 Newington Road,	1902
	Dr J. Stuart Ross, 15 Lennox Street,	1908
205	Dr William Russell, 3 Walker Street,	1884
	Dr E. G. Salt, 59 George Square,	1895
	Dr James Scott, 43 Minto Street,	1895
	Dr Theodore Shennan, 95 Mayfield Road,	1897
	Dr W. C. Sillar, 2 Lockharton Gardens,	1900

		Date of Admission.
210	Emeritus Professor Sir Alexander R. Simpson, 52 Queen Street,	1859
	Dr F. D. Simpson, 9 Hampton Terrace,	1905
	Dr G. F. Barbour Simpson, 43 Manor Place,	1900
	Dr John W. Simpson, 19 Lansdowne Crescent,	1901
	Dr Arthur H. H. Sinclair, 5 Walker Street,	1900
215	A. A. Scot Skirving, Esq., C.M.G., 8 Randolph Crescent,	1897
	Dr A. T. Sloan, 22 Abercromby Place,	1885
	Dr James Smith, 4 Brunton Place,	1891
	Dr J. Cormack Smith, 9 Brunton Place,	1896
	Dr J. W. Steven, 13 Manor Place,	1900
220	Dr John Stevens, 78 Polwarth Terrace,	1892
	Dr Norman L. Stevenson, 7 Alva Street,	1902
	Dr Ian Struthers Stewart, 13 Castle Terrace,	1906
	Dr J. S. Stewart, 15 Merchiston Place,	1885
	Dr Robert Stewart, 25 George Square,	1892
225	Dr William Stewart, 146 Ferry Road, Leith,	1889
	H. J. Stiles, Esq., 9 Great Stuart Street,	1889
	Dr John Strachan, Dollar,	1867
	John W. Struthers, Esq., 2 Chester Street,	1901
	W. J. Stuart, Esq., 9 Chester Street,	1902
230	Dr William G. Sym, 12 Alva Street,	1889
	Dr W. Taylor, 12 Melville Street,	1871
	Dr W. Macrae Taylor, 8 Melville Street,	1903
	Dr C. H. Thatcher, 8 Melville Crescent,	1876
	Dr L. H. F. Thatcher, 8 Melville Crescent,	1910
235	Dr R. Thin, 25 Abercromby Place,	1890
	Professor Alexis Thomson, 39 Drumsheugh Gardens,	1887
	Dr H. Torrance Thomson, 3 Hillside Crescent,	1900
	Dr J. C. Thomson, Dumfries.	1910
	Dr John Thomson, 14 Coates Crescent,	1887
240	Dr George Thyne, 13 Brunswick Street,	1904
	Dr T. J. Thyne, 16 Randolph Crescent,	1898
	Dr John Tod, 67 Ferry Road, Leith,	1895
	Dr H. M. Traquair, 30 Walker Street,	1910
	Sir John Batty Tuke, M.P., 20 Charlotte Square,	1864
245	Dr F. J. Turnbull, 6 Randolph Place,	1896
	Dr Dawson Turner, 37 George Square,	1890
	Dr Logan Turner, 27 Walker Street,	1892
	Henry Wade, Esq., 6 Manor Place,	1903
	Dr Alexander Walker, 1 Lauder Road,	1907
250	Dr Norman Walker, 7 Manor Place,	1891
	D. Wallace, Esq., C.M.G., 29 Charlotte Square,	1887
	Dr Chalmers Watson, 11 Walker Street, *Editor of Transations*,	1894
	Dr W. de Watteville, Kingussie,	1902
	Dr A. D. Webster, 18 Minto Street,	1883
255	D. P. D. Wilkie, Esq., 56 Manor Place,	1909
	Dr James Wilson, 53 Inverleith Row,	1900
	J. L. Wilson, Esq., 4 Buccleuch Place,	1883
	Dr A. Murray Wood, 180 Ferry Road, Leith,	1907
	Dr Russell E. Wood, 9 Darnaway Street,	1883
260	Professor John Wyllie, 44 Charlotte Square,	1868

(*b*) Members Exempted under Rule V. from paying the Annual Subscription.

		Date of Admission.
	Dr D. Aitken, 152 *Packington Street, Islington, London, N.*,	1887
	Dr H. de M. Alexander, *Aberdeen District Asylum, Aberdeen*,	1901
	Dr D. H. Anderson, 143 *Abbey Road, Barrow-in-Furness*, .	1887
	Dr John Anderson, *Pitlochry*, ...	1896
265	Dr J. A. Armitage, 58 *Waterloo Road, Wolverhampton*, .	1887
	Dr F. L. Atkinson, *Victoria Road, Devonport, Auckland, New Zealand*, ...	1904
	Dr W. Badger, *Penicuik*, ...	1882
	Dr Edwin Baily, *Oban*, ...	1883
	Dr Andrew Balfour, *Khartoum*, ...	1895
270	Dr J. H. Balfour, *Portobello*.	1881
	Dr Alexander Ballantyne, *Dalkeith*, ...	1872
	Dr Noel D. Bardswell, *Midhurst Sanitorium, Sussex*, .	1901
	Dr W. H. Barrett, 29 *Park Crescent, Hesketh Park, Southport*,	1890
	Sir George T. Beatson, K.C.B., 7 *Woodside Crescent, Glasgow*,	1895
275	Professor James M. Beattie, M.B., *The University, Sheffield*,	1904
	Surgeon-Captain C. H. Bedford, *Bengal Army, care of W. Watson & Co.*, 28 *Apollo Street, Bombay*, . .	1889
	Dr Charles Begg, 62 *Pulteney Street, Bath*, . . .	1900
	Dr Benjamin D. C. Bell, *Kirkwall*, ...	1889
	Dr G. J. H. Bell, *Bengal Army*, ...	1884
280	Dr J. H. Montgomerie Bell, *Brunswick Square, Camberwell*,	1905
	Dr G. H. Bentley, *Kirkliston*, ...	1877
	Professor Richard J. A. Berry, M.D., *University of Melbourne*,	1893
	Dr J. S. Beveridge, *Melton Constable, Norfolk*, . .	1861
	Dr J. W. Black, 15 *Clarges Street, Piccadilly, London, W.*, .	1865
285	Dr D. G. Braidwood, *Halkirk, Caithness*, . . .	1889
	Dr Andrew Brown, 1 *Bartholomew Road, Kentish Town, London, N.W.*, ...	1884
	Dr H. Grey Brown, 47 *Evan Street, Stonehaven*, . .	1905
	Dr James Murdoch Brown, ...	1885
	Dr John Brown, ...	1878
290	Dr J. Macdonald Brown, 2 *Fragnall, Hampstead, London, N.W.*, ...	1883
	Dr William Brown, *Braemar*, ...	1904
	Dr Lewis C. Bruce, *The Asylum, Murthly, Perthshire*, .	1895
	Dr William Hartley Bunting, 22 *Islington Row, Edgbaston, Birmingham*, ...	1900
	Dr Malcolm Campbell, *Brechin*, ...	1904
295	Dr D. A. Carruthers, 5 *Charlotte Street, Perth*, . .	1901
	Dr S. W. Carruthers, 44 *Central Hill, Norwood, London, S.E.*,	1899
	Professor Cleland, *The University, Glasgow*, . . .	1864
	Dr A. R. Coldstream, 24 *Lung Arno Nuovo, Florence, Italy*,	1878
	Dr William Craig, *Cowdenbeath, Fife*, . . .	1894
300	Dr J. R. Crease, 2 *Ogle Terrace, South Shields*, . .	1885
	Dr J. Frank Crombie, *North Berwick*, . . .	1897
	Dr Bampylde Daniell, *Adderley Street, Cape Town*, . .	1903
	Dr P. M. Deas, *Wonford House, Exeter*, . . .	1868
	Dr Frederick Dittmar, 72 *Murrayfield Gardens*, . .	1906
305	Dr A. Home Douglas, 5 *Rue de Russie, Nice, France*, .	1889
	Dr J. J. Douglas, 42 *Central Hill, Upper Norwood, London*, .	1891
	Dr C. C. Easterbrook, *Crichton House, Dumfries*, . .	1899
	Dr J. W. Eastwood, 18 *Farndon Road, Oxford*, . .	1871
	Dr F. A. Elkins, *The Asylum, Leavesden, near King's Langley*.	1893

		Date of Admission
310	Dr R. W. Felkin, 12 *Oxford Gardens, North Kensington, London, W.*,	1885
	Dr A. N. Fell, 11 *Lexden Road, Colchester*,	1907
	Dr Samuel Fergusson, *Lylestone House, Alloa*,	1902
	Right Hon. Sir R. B. Finlay, K.C., LL.D., *Middle Temple, London*,	1864
	Dr Charles Forsyth, *Alexandra Buildings, Hong-Kong*,	1903
315	Dr Fourie, *South Africa*,	1908
	Dr Simson C. Fowler, *Juniper Green*,	1892
	Dr F. W. Foxcroft, 47 *Newhall Street, Birmingham*,	1893
	Dr Dyce Fraser, 163 *Cornwall Road, London, S.W.*,	1883
	Dr William Gayton, 11 *Redbourne Avenue, North Finchley, London, N.*,	1886
320	Dr Campbell Geddes, Professor of Anatomy, *College of Surgeons, Dublin*,	1904
	Dr Rae Gibson,	1910
	Dr T. Paul Gray, 94 *Adelaide Road, Hampstead, N.W.*,	1899
	Dr W. C. Greig, *Otago, New Zealand*,	1884
	Dr George K. Grimmer, *Canada*,	1897
325	Dr Groesbeck, *Cincinnati*,	1875
	Dr W. B. T. Gubbin, *Redland Grove, Bristol*,	1893
	Dr John Haddon, *Denholm, Roxburghshire*,	1883
	Dr Archibald Hall, *Montreal*,	1853
	Dr J. W. Hamp, *Penn Road, Wolverhampton*,	1887
330	Dr John Hardie,	1895
	Dr A. W. Hare, *Newquay, Cornwall*,	1883
	Dr J. Home-Hay, *Alloa*,	1880
	Professor J. Berry Haycraft, *Llanishen, Cardiff*,	1889
	Dr Stanley Haynes, *Malvern, Worcestershire*,	1864
335	Dr R. Dundas Helm, 13 *Portland Square, Carlisle*,	1892
	Dr David Hepburn, Professor of Anatomy, *University College, Cardiff*,	1902
	Dr R. E. Horsley, *Crail, Fife*.	1886
	Dr James Hunter, *St Catherine's, Linlithgow*,	1890
	Dr W. Hunter, 103 *Harley Street, Cavendish Square, London, W.*,	1887
340	Dr Robert Inch, *Gorebridge*,	1887
	Dr James Johnston, 53 *Princes Square, Bayswater, London, W.*,	1871
	Dr J. Carlyle Johnstone, *The Asylum, Melrose*,	1882
	Dr J. Keay, *Bangour Asylum, Uphall*,	1887
	Dr A. J. Keiller, *New Brunswick, Canada*,	1889
345	Dr George Keith, *Moidart Cottage, Currie*,	1845
	Dr Skene Keith, 58 *Upper Berkeley Street, Portman Square, London, W.*,	1885
	Dr W. Scott Lang,	1868
	Dr J. W. Leech, 12 *Eldon Square, Newcastle-on-Tyne*,	1901
	Professor R. F. C. Leith, M.B., *The University, Birmingham*,	1886
350	Dr John Linton, *Aberdour*,	1863
	Dr G. R. Livingston, 47 *Castle Street, Dumfries*,	1901
	Dr T. Davy Luke, *Peebles*,	1900
	Dr Donald Macaulay, *Jumpers' Gold Mining Co., P.O. Box 1043, Johannesburg*,	1895
	Dr Stuart M'Donald, *Newcastle-on-Tyne*,	1901
355	Dr John A. Macdougall, *Cannes, France*,	1875
	Dr William C. M'Ewan, *Prestonpans*,	1892
	Professor Sir J. M'Fadyean, 101 *Great Russell Street, London, W.C.*,	1888
	Dr F. Reid Mackay, *New Zealand*,	1899
	Dr John Mackay, *Aberfeldy*,	1881
360	Dr W. B. Mackay, 23 *Castlegate, Berwick-on-Tweed*,	1893
	Professor M'Kendrick, *Maxie Burn, Stonehaven*,	1870

		Date of Admission.
	Dr James Mackenzie, 17 *Bentinck Street, London*,	1894
	Dr Robert Mackenzie, *Napier, Nairn*,	1893
	Dr Theodore C. Mackenzie, *The Asylum, Lochgilphead*,	1904
365	Dr William A. Mackintosh, 3 *Pork Terrace, Stirling*,	1896
	Dr William MacKirdy, 157 *Lincoln Road, Peterborough*,	1907
	Dr David W. Maclagan, *care of Dr Harrison, Eltham, Taranaki, New Zealand*,	1901
	Dr J. Shaw M'Laren, *India*,	1887
	Dr Roderick M'Laren, 23 *Portland Square, Carlisle*,	1882
370	Dr J. C. G. Macnab, *South Africa*,	1904
	Dr Alex. H. Macpherson,	1901
	Dr G. Douglas M'Rae, *Glengall House, Ayr*,	1904
	Dr William B. MacTier, 7 *Queen's Gardens, St Andrews*,	1901
	Dr Samuel Macvie, *Chirnside*,	1897
375	Dr J. W. Martin, *Charterhall, Newbridge, Dumfries*,	1888
	Dr J. B. Mears,	1907
	Dr Andrew W. Messer, *Lemington, Scotswood-on-Tyne*,	1899
	Dr James Middlemass, *Boro' Asylum, Ryhope, Sunderland*,	1895
	Dr E. A. Mills-Roberts, *Bethesda, Bangor*,	1897
380	Dr J. Moolman, *Cape of Good Hope*,	1877
	Dr E. C. Moore,	1892
	Dr J. E. Moorhouse, 6 *Melville Terrace, Stirling*,	1901
	Dr A. E. Morison, *Mowbray Villa, Sunderland*,	1889
	Dr J. Rutherford Morison, 14 *Saville Row, Newcastle-on-Tyne*,	1882
385	Professor Robert Muir, *The University, Glasgow*,	1894
	Dr D. G. Macleod Munro, 1 *Oriel Villas, Cheltenham*,	1898
	Dr Peter Murray, *Durie Street, Leven*,	1904
	Dr W. H. Myers, *Indiana, U.S.A.*,	1906
	Dr Leith Napier, *The General Hospital, Adelaide, South Australia*,	1879
390	Dr Ernest F. Neve, *Srinagar, Kashmir, India*,	1884
	Dr Peter Orphoot, *North Berwick*,	1865
	Dr T. Wyld Pairman, *Hinemoa, Moffat*,	1884
	Professor Bell Pettigrew, *St Andrews*,	1873
	Dr Alexander Peyer, *Zürich*,	1893
395	Dr J. A. Philip, *Rue Victor Hugo, Boulogne-sur-Mer, France*,	1878
	Dr J. H. Pringle, 172 *Bath Street, Glasgow*,	1886
	Dr S. Hale Puckle, *Bishop's Castle*,	1885
	Dr R. B. Purves, 5 *Minster Yard, Lincoln*,	1899
	Dr J. Maxwell Ross, *St Ruth's, Maxwelltown, Dumfries*,	1882
400	Dr J. R. Home Ross, *Burmah*,	1888
	Dr Joseph C. Ross, *Withington*,	1884
	Dr S. Rumboll, *Cavendish House, Leeds*,	1887
	Dr Thomas Russell, *Beechwood, Parkhead, Glasgow*,	1888
	Dr Gordon Sanders, *Cannes, France*,	1892
405	Dr F. A. Saunders, *Grahamstown, South Africa*,	1884
	Dr Thomas R. Scott, *Musselburgh*,	1884
	Dr W. Vernon Shaw, 42 *Yorkersgate, Malton, Yorks*,	1905
	Dr Herbert D. Shepherd, 45 *Kedleston Road, Derby*,	1903
	Dr G. F. Shiels, 229 *George Street, San Francisco*,	1887
410	Dr W. H. Shirreff, *Port Fairy, Victoria, Australia*,	1883
	Dr William Simmers, *Gordon, Berwickshire*,	1894
	Dr Herbert Sloman, 65 *Goldington Road, Bedford*,	1906
	Dr John Smith, *Brycehall, Kirkcaldy*,	1889
	Dr William Smith, *America*,	1890
415	Dr W. Ramsay Smith, *The General Hospital, Adelaide, South Australia*,	1892
	Dr Van Someren, *Northwood, Middlesex*,	1845
	Dr J. W. Somerville, 12 *Abbotsford Road, Galashiels*.	1905
	Dr Arthur J. de Spiganovicz, *Dunloe, Tranent*,	1905
	Dr A. M. Stalker, 156 *Perth Road, Dundee*,	1893
420	Dr Graham Steell, 96 *Moseley Street, Manchester*,	1877

		Date of Admission.
	Professor Stephenson, *University*, *Aberdeen*, . . .	1861
	Dr John Stevenson, ...	1900
	Dr Robert Stirling, 4 *Atholl Place*, *Perth*, . . .	1891
	Professor Ralph Stockman, *The University*, *Glasgow*, .	1891
425	Dr H. R. Storer, *Newport*, *Rhode Island*, *U.S.*, . .	1855
	Dr John Struthers, *Ngamakwe*, *Transkei*, *South Africa*, .	1895
	Dr J. F. Sturrock, *Arima*, *Broughty Ferry*, . . .	1887
	Professor J. Symington, *Queen's College*, *Belfast*, . .	1878
	Dr C. C. Teacher, *North Berwick*, ...	1887
430	Dr C. Templeman, *Sanitary Office*, *Bell Street*, *Dundee*, .	1891
	Dr J. A. Douglas Thompson, *Comberton House*, *Halesowen*, *Manchester*, ...	1901
	Dr J. Stitt Thomson, *Castle Hill House*, *Lincoln*, . .	1877
	Dr J. Batty Tuke, Jr., *Mavisbank*, ...	1886
	Dr R. S. Turner, *Keith*, ...	1867
435	Dr T. Edgar Underhill, *Dunedin*, *Burnt Green*, *Worcestershire*, ...	1885
	Dr A. R. Urquhart, *Murray House*, *Perth*, . . .	1893
	Dr Gopal Govind Vatve, *care of H.H. the Rajah of Miraj*, *Bombay*, *India*, ...	1895
	Dr David Waterston, F.R.C.S.E., *London*, . . .	1900
	Dr Walter de Watteville, *Kingussie*, ...	1902
440	Dr J. H. Douglas Webster, ...	1907
	Dr A. B. Whitton, *Aberchirder*, ...	1886
	Dr H. Douglas Wilson, 3 *Rippon Road*, *Harrogate*, . .	1904
	Professor G. Sims Woodhead, *Pathological Laboratory*, *New Museums*, *The University*, *Cambridge*, . . .	1883
	Dr Yellowlees, 6 *Albert Gate*, *Dowanhill*, *Glasgow*, . .	1862

N.B.—*Members are requested to communicate with the Secretaries if they discover any errors or omissions in the List, and also to intimate all changes in their addresses.*

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TRANSACTIONS

OF THE

MEDICO-CHIRURGICAL SOCIETY

OF EDINBURGH

TRANSACTIONS

OF THE

MEDICO-CHIRURGICAL SOCIETY OF EDINBURGH

FOR SESSION LXXXIX, 1910-11

Meeting I.—November 2, 1910

DR BYROM BRAMWELL, *President, in the Chair*

I. ELECTION OF OFFICE-BEARERS

The following gentlemen were elected office-bearers:—*President*, Dr Byrom Bramwell; *Vice-Presidents*, Dr W. Allan Jamieson, Mr J. M. Cotterill, and Mr Charles W. Cathcart; *Council*, Dr James Ritchie, Mr David Wallace, Dr J. S. Fowler, Dr John Cumming, Dr T. J. Thyne, Dr Edwin Bramwell, Mr J. W. Struthers, Dr R. J. Johnston; *Treasurer*, Mr J. W. Dowden; *Secretaries*, Dr Francis D. Boyd, C.M.G., and Mr Alexander Miles; *Editor of Transactions*, Dr Chalmers Watson.

II. ELECTION OF MEMBERS

James Miller, M.D., F.R.C.P. Ed., 24 Queen Street, and John Christopher Thomson, M.A., M.D., D.P.H., D.T.M. & H. Camb., 6 Morningside Road, were elected ordinary Members of the Society.

III. Treasurer's Annual Statement

EDINBURGH MEDICO-CHIRURGICAL SOCIETY'S ACCOUNTS

FOR SESSION 1909-1910.

INCOME				EXPENDITURE			
Entrance Fees	£7 7 0			Turnbull & Spears, printing Vol. XXVIII. of Transactions, Postage, etc.	£72 0 7		
Annual Subscriptions	125 10 0			Rent of Hall	10 0 0		
Annual Subscriptions, arrears	1 10 0			Collector	4 0 0		
Subscriptions for Transactions	0 15 0			Neill & Co., Billets, Postage	25 6 0		
Sale of Transactions, Vols. 26 and 27	8 14 0			Alex. Frazer, Microscopes, etc.	5 8 6		
Caledonian Railway Debenture Stock, Dividends, November 1909 and May 1910	13 3 8			A. & D. Padon, Stationery	4 18 2		
Dominion of Canada 3½% Stock Dividends, January 1910 and July 1910	9 17 10			Typing, Gardiner & M'Adam	2 16 3		
	£166 17 6			Waterston & Sons, Minute-Book	0 10 6		
				J. M'Callum, Tea and Coffee	6 0 0		
				Doorkeeper	1 6 0		
				Fenton, Russell & Co. (Craig Testimonial)	25 10 0		
				Petty Cash	0 1 11		
					£157 17 11		
Balance in Bank, November 1, 1909		48 1 1		Balance in Bank, November 1, 1910		57 0 8	
		£214 18 7				£214 18 7	

STATE OF FUNDS.

At November 1909.				At November 1910.			
Caledonian Railway Debenture Stock (Purchase Price)	£484 11 6			Caledonian Railway Debenture Stock (Purchase Price)	£484 11 6		
Dominion of Canada 3½% Stock (Purchase Price)	298 2 6			Dominion of Canada 3½% Stock (Purchase Price)	298 2 6		
Balance in Bank	48 1 1			Balance in Bank	57 0 8		
	£830 15 1				£839 14 8		

J. W. DOWDEN, *Hon. Treasurer.*

We have examined the accounts and compared them with the vouchers, and have found them all correct.

GEORGE HUNTER.
W. G. AITCHISON ROBERTSON.

November 17th, 1910.

IV. Exhibition of Patients

1. *Mr Cotterill* and *Dr Bruce* showed—

Patient with ACQUIRED HYDROCEPHALUS cured by opening of foramen of Magendie.

Attack of acute cerebral meningitis in 1908, followed by recurrent headaches, vomiting, gradual impairment of sight, optic neuritis—Abolition of left abdominal reflexes—Recurring losses of control of bowels and bladder, then extreme weakness, first of left leg, then of right—Increase of stuperose condition—Blindness complete—Separation of sutures by distension of brain ventricles—Operations by Mr Cotterill: (1) puncture of lateral ventricles; (2) removal of large portion of occipital bone, reopening of foramen of Magendie, which had been closed by fibrous tissue—Result, gradual and practically complete recovery of motor power, sight, intelligence, and control of reflexes.

2. *Mr Miles* showed—

Patient after removal of Gasserian ganglion for TRIGEMINAL NEURALGIA.

Wm. F., æt. 60—Woolsorter—Symptoms of neuralgia for over five years, gradually getting worse—For two years unable for work—Typical clinical picture of tic douloureux—Operation, 17th June 1910—Gasserian ganglion, with second, third divisions of trigeminal nerve, removed by modified Hartley-Krause operation—Pain immediately relieved.

3. *Dr Boyd* showed—

Patient after CEREBRO-SPINAL MENINGITIS, possibly of tuberculous origin.

Patient comatose with squint—Head retraction—Vomiting—Early optic neuritis—Spinal fluid purulent—Von Pirquet's reaction positive—No micro-organisms found in cerebro-spinal fluid—Treatment, repeated lumbar puncture and collargol injections—Recovery.

4. *Dr Byrom Bramwell* showed—

A case of NERVOUS DISEASE OF DOUBTFUL NATURE FOR DIAGNOSIS. The patient, a male, aged 20, single, a painter, was admitted to the Edinburgh Royal Infirmary on 10th October 1910, complaining of weakness in the legs and weakness and stiffness of the left arm.

He stated that the condition had commenced without obvious cause a year previously. He first felt the left leg weak, and four

months afterwards the left arm became involved. There was no history of lead poisoning. The *family history* was good, no other members having been affected with any form of nervous disease.

On examination, both legs were found to be weak, especially the left; the left arm was weak; there was marked rigidity of both pectorals and lattissimus dorsi muscles, and some rigidity of the left elbow; the feet were in a condition of pes cavus, resembling the condition which is met with in Friedreich's ataxia. The small muscles of the feet and hands were wasted, especially those of the left hand. The left forearm muscles were also wasted and, to some extent, the deltoids. No fibrillary twitchings were detected in any of the affected muscles, and there was no reaction of degeneration. The knee-jerks were markedly exaggerated; there was double ankle-clonus and a double extonsor response. The deep reflexes in the left upper extremity were exaggerated. On exposure to air the feet and hands get blue and cold. The bladder and rectum were unaffected. On looking to the right there were slight nystagmus. The sensory functions were practically normal. On the back of the arms and front of the legs, both above and below the knee, tactile sensation was said to be delayed. The general health was good.

5. *Dr G. A. Gibson* showed—

A case of CHRONIC POLIO-ENCEPHALITIS INFERIOR.

Man, aged 57; served in youth in the 17th Lancers, and afterwards engaged in manual labour; acquired lues when in the army; a few months ago began to have trouble in speech and swallowing. Now he has atrophy and fibrillation of tongue, and feebleness of lips, with tendency to escape of fluids through nose; speech is very imperfect, both articulation and phonation being impaired. The anterior nuclei seem intact, and the case is one of chronic polio-encephalitis inferior.

V. ORIGINAL COMMUNICATIONS

1. EPITHELIOMA OF THE TONGUE—A REVIEW OF SIXTY HOSPITAL CASES.

By F. M. CAIRD,
Regius Professor of Clinical Surgery, Edinburgh

THERE are sites in the human body wherein carcinoma assumes a specially intractable character, and where the most far-reaching efforts of the surgeon to attain a radical cure are extremely dis-

appointing in their results. We may contrast in this respect the larynx and the tongue, the sigmoid flexure of the colon and the rectum. Carcinoma of the larynx confined within a cartilaginous box, and carcinoma of the sigmoid flexure enveloped by its protecting envelope of peritoneum, resemble each other in so far that a relatively favourable prognosis can be given as to the ultimate result of operation, while, on the other hand, carcinoma of the tongue and carcinoma of the rectum are alike more uncertain as to the ultimate issue of surgical intervention. In the latter sites the anatomical relations of the muscular planes involved, the free lymphatic connections, and the lack of a good defensive environment all favour rapid dissemination of the disease. It is notorious that our treatment of lingual carcinoma, frequent as the malady is, easily recognised, and that, too, at an early date, cannot be said, as yet, to rank amongst the brilliant triumphs of surgery.

On this account a review of sixty cases under our care during the last ten years in the Royal Infirmary may prove instructive. The list appreciably loses in value from the somewhat meagre nature of some of the clinical records and from the difficulty experienced in tracing the after-history of cases drawn from a floating population, but it at least represents an individual experience. Speaking generally, the disease is peculiar to the male sex, rarely attacking women. It begins as a local induration, or a hard elevated area, on the lateral border of the tongue, where there may already exist a crack or fissure. The covering epithelium gives way, an ulcer forms, and a most characteristic pain, referred to the ear and to the temporal region, annoys the patient, so that he frequently appears with cotton-wool plugging the ear. Enlargement of the lymphatic cervical glands speedily ensues. If the trouble be situated near the root of the tongue, or if it spread to the floor of the mouth, the greater is the inconvenience felt by the patient in the form of discomfort or pain when chewing, swallowing, and speaking. There may be a copious flow of saliva, and the breath acquires an offensive odour. Difficulty in protruding the tongue is experienced, according to the fixation and involvement of the organ.

Antecedent irritation has long been recognised as at least predisposing to the development of carcinoma. The laity are prone to cite local irritation of some kind, chiefly that from carious or jagged teeth, as the main source of this malady.

Of our sixty cases twenty-seven ascribed the disease to this cause, although one patient was convinced that induration appeared prior to the dental irritation; eighteen already had the offending tooth extracted, and thereby thought some amelioration of pain ensued; four had the projecting angles of the tooth filed smooth; seven came with the offending tooth *in situ*; three blamed a badly-fitting artificial denture; two were satisfied that the malignant ulcer arose at a point facing that where they held the tobacco pipe; three had made a rather prolonged use of caustics for apparently simple conditions; and one had scalded her mouth with porridge eight weeks before the epithelioma developed. Thus thirty-eight gave a definite account of an antecedent local irritation.

Tobacco has been branded as a possible factor. Amongst the fifty-four male cases it is interesting to find that no one is chronicled as a non-smoker. The pipe was used by twenty in moderation, eleven to excess (over two and a half ounces a week); details are lacking as to the others. The habit of smoking was abandoned as the disease advanced. One patient occasionally obtained relief by smoking, but as frequently the reverse.

In regard to *alcohol* three patients were total abstainers, fourteen drank to excess, eight in moderation, and of fifteen there is no definite information.

Syphilis.—In no patient were syphilitic manifestations discovered, and only four males acknowledged infection.

Leucoplakia is only noted three times as occurring on the tongue and cheeks.

Nineteen patients could give no history of any antecedent irritation, two were edentulous, and one, a Highlander, 70 years of age, had a perfectly sound set of teeth.

A *hereditary history of carcinoma* was elicited from five patients. On the paternal side a death from cancer of the stomach; on the maternal, two cases of mammary cancer, one of the œsophagus, and one of the pelvic organs.

Of the six females three blamed jagged teeth, one irritation from a tooth-plate, and one, as already stated, had scalded her tongue with porridge.

A consideration of the above points tends to confirm us in the relationship which obtains between antecedent irritation and malignant disease. It also urges us to enter a plea for the same

care of the teeth in the later years of adult life that has been so strongly advocated in early youth. One would emphasise the apprehension with which the surgeon views the supposed "tooth-aches and earaches" of mature years, and we would draw the attention of the dentist to the importance of investigating the state of the tongue in such patients.

The question of diagnosis need not detain us. It rarely presents any difficulty, and microscopic examination is so easily carried out that it should be routine in every case to confirm an opinion or to clear up a doubt. For this purpose the ulcerated area is cleansed, and a small portion is snipped from out the base of the ulcer, remote from its epithelial edge. This is teased out and stained with picro-carmine, or it may be more satisfactorily investigated by cutting in series with a microtome. The latter method is needful in doubtful cases. Few pathological conditions are so clearly revealed, and give such a positive result on microscopic examination. On two occasions only have we failed to obtain a definite result. Curiously enough both patients were females. The first of the two may have had a sarcoma. The histories are as follows :—

I. Miss A. H., æt. 60, for the last eighteen months has had enlarged lymphatic glands on the right side of the neck. Four months ago she caught cold, the glands increased in size, and her tongue became swollen towards the back. Her false teeth now rubbed on the tongue, caused a slight abrasion, so she removed them. A growth next appeared at the irritated site, steadily increased, and occupied the greater part of the right side of the tongue. It rose from a broad base above the level of the dorsum, was covered with a yellowish exudate, and was of firm but not indurated consistence. The lymphatic glands on each side of the neck were firm, mobile and tender. Latterly she experienced pain on the right side of the face. The pathologist reported that the part removed for examination consisted almost wholly of granulation tissue, there was nothing distinctly epithelial to be made out. Notwithstanding this it was thought right to remove the affected half of the tongue. The glands thereafter subsided, and the patient made an excellent recovery. We learned that the patient died five months after from fungating cervical glands.

II. Mrs W., æt. 39. Since her fifteenth year she has been unable to swallow properly. She has also been subject to sore places on the left side of the tongue and cheek, the mucosa

becoming red, ulcerating, and again rapidly healing. For the last three months the left side of her tongue has been painful, as also the side of the neck and face. The tongue showed a superficial glossitis on the left side, and somewhat remote posteriorly a thickened epithelial patch, not ulcerated nor hard. On two occasions pieces of tissue removed from this were free from any evidence of epithelioma. The patient was therefore sent home with instructions to use a mouth wash, and to report progress. She returned after six months. There was now advanced epithelioma of tongue and glands, confirmed at once on microscopic examination, and the condition was inoperable.

Even if we admit a single error in regard to the microscopic features, as revealed in the first examination of the above case, it in no way detracts from the great practical value of such a method of investigation, a method so trustworthy and important that we cannot afford to dispense with its use in any case.

The only treatment for lingual carcinoma which at present holds out prospect of hope is that by the knife. No other is of any avail. The so-called radical measures generally constitute a two-stage operation. We are indebted to Mr Butlin, who brought our operative procedure into line with those practised in other regions of the body when he advocated a systematic attack upon the adjacent lymphatic areas. The first part consists in removal of the half or the whole tongue after the manner of Whitehead or of Syme. The second, about ten days after, is Butlin's dissection of the side of the neck affected, from the angle of the jaw to the sterno-clavicular articulation, and requires complete removal of the connective tissue exposed, including the platysma, lymphatics, submaxillary, and portions of the parotid salivary glands, and even portions of the internal jugular vein, etc., if need be. Such sweeping measures are accompanied by a remarkably low mortality, so low, indeed, that, despite uncertainty as to the ultimate result, we are justified in urging operation. The chances of a cure are not, indeed, so probable as those which follow upon pylorectomy for gastric carcinoma, but the risk incurred is not nearly so great. Apart from the usual surgical precautions, the vascularity of the regions involved and the opportunities for free drainage diminish the danger. Moreover, local recurrence in the mouth is comparatively seldom. The patient's life is prolonged, and he is spared, at but small danger, the misery and suffering associated with a foul, painful mass in the mouth, and the frequent glandular return

of the disease in the neck is a less trying termination to life. Hence partial operations in which the primary lingual disease alone is removed, and the secondary glandular implication is left untouched, may be perfectly justifiable and, indeed, welcome.

Certain circumstances lead to modifications in the method of operation. For several years we have been accustomed to perform the first stage of the operation under local anæsthesia, and, we have reason to believe, with great advantage. The patient's mouth having been duly prepared, he lies on the table, and the tip of the tongue is infiltrated with a half per cent. solution of cocain to which a few drops of adrenalin have been added. Two stout silk loops are then carried through the tongue on each side of the mid-line half an inch from the tip; the tongue is pulled forward, and its frenum, the mid-line, the lateral and basal attachments are freely infiltrated with the solution. Within five minutes the operation of Whitehead may be performed with knife or scissors in any easy fashion, for, thanks to the infiltration, connective tissue planes are opened up, anatomical details and the vessels are rendered clearly visible. The halves of the tongue are completely under control owing to the silk loops, no gag is required, and the patient may aid the surgeon by rinsing out the mouth if required. Even in cases where the jaw has to be divided, as in Syme's operation, we may inject the solution into the periosteum, extract incisors, and sever the bone with a Gigli saw, and that in such a painless manner that the patient neither raises his hand nor makes any protest. We endeavour to remove the entire half of the organ, leaving only the geniohyoid and the mylohyoid muscles. Should the disease extend to the alveolar margin we do not remove the whole depth of the bone. A free excision, leaving the hard sclerosed lower border, thus preserving an intact mandibular arch, is of marked advantage, and the dentist can thereafter fit an excellent prosthetic apparatus if his services have been employed prior to operation. We have not found recurrence in the bar of bone so preserved. One is much impressed by the painless character and by the slight discomfort of such an operation performed under local anæsthesia, as also by the ease with which the patient speaks and feeds immediately after. We prefer cocain to novocain in tongue cases, because of the rapidity of its action and the fact that so much of the drug escapes during operation. There is one associated disadvantage, namely, after-oozing of blood. Every bleeding point should therefore be secured. The patient, moreover, sits up at

once in bed, and by making free use of the douche rapidly checks bleeding. We treat the raw surface with iodoform and boric powder, leaving it freely open, although the method of using catgut stitches to diminish the extent of the wound might further act in diminishing reactionary hæmorrhage.

It will be found that the adjuvant laryngotomy or tracheotomy, so strongly advocated by many surgeons, is quite unnecessary when local anæsthesia is employed.

The second stage of the operation requires a general anæsthetic. It is better to remove the affected portions of the tongue before the glands, otherwise it is conceivable that lymphatic spread to the other side of the neck might be favoured when the normal channels of the affected area are cut off. At this second stage the oral cavity may be opened into, and as this entails sepsis in the upper regions of the incision free drainage is essential. We have generally removed the facial artery and vein *en masse* along with the contents of the sub-maxillary triangle.

The average age of the patients was forty-nine years, the extremes being twenty-three and seventy-three. Thirteen cases were inoperable. The annexed table of forty-seven submitted to operation is in chronological order, and is therefore the more instructive. As indicated, the type of operation conforms more or less to the methods associated with the names of Syme, Whitehead and Butlin. There were seven fatal cases, all, however, exceptionally severe, requiring ablation of the entire tongue, tonsils, portions of the pharyngeal wall and full depth of the jaw; in each a general anæsthetic had been administered.

It will be noted that only sixteen patients at present survive. Ten, twelve months since operation, and one each, twenty months, twenty-four months, thirty months, thirty-one months, seven years and eight years. Fourteen died within a year after operation, and others each within sixteen, twenty-eight and thirty-six months. Eight cannot be traced.

Such results are apt to make one despond as to the prospects of a radical cure. Even in cases where the microscope did not reveal any trace of malignancy in the lymphatic glands removed a return has taken place. However, it must be admitted that results in private practice are rather more encouraging, probably because the patients apply at an earlier date. Moreover, there is good reason to believe that the still more extensive operation in which the diseased area, including the sterno-mastoid, is removed *en*

bloc, as suggested by Maitland and by Crile, will yet afford a greater measure of success.

For a list of literature see the excellent article on "Carcinoma of the Mouth and Tongue," by Collins Warren, *Annals of Surgery*, 1908.

Name, Age, Date, Duration of Disease.	Antecedent Irritation.	Anæsthetic. Operation. Parts Removed.	Microscopic Report on— 1. Tongue. 2. Glands.	Result of Operation.	Further History.
1. Mr C. A. 73. 22/8/00. 9 months.	None.	Chloroform. Whitehead. Left half and alveolar margin of jaw.	1. Epithelioma.	R.	Hale and well 8/5/08. Not traced since.
2. Mr G. H. 55. 19/1/01. 3 months.	None.	Chloroform. Syme. Left half and removal of glands.	1. ,,	R.	Died one year after.
3. Mr J. W. 62. 21/1/01.	Tooth.	Chloroform. Whitehead. Left half.	1. ,,	R.	Recurrence, glands removed, 4/5/01. Not traced.
4. Mr W. A. 63. 1/9/02.	Tooth.	Chloroform. Whitehead. Right half and glands.	1. ,,	R.	Died Oct. 1903.
5. Mrs B. 23. 25/10/02. 7 weeks.	Tooth.	Chloroform. Whitehead. Right half and glands, internal jugular vein.	1. ,,	R.	Died within a year.
6. Mr A. Y. 51. 7/7/03. 7 months.	Tooth.	Chloroform. Ligature lingual artery and glands. Whitehead. Right half.	1. ,,	R.	Died 1/4/04.
7. Mr T. C. 44. 26/9/03. 7 weeks.	Tooth.	Chloroform. Ligature lingual artery and glands. Whitehead. Right half.	1. ,, 2. Glands normal.	R.	Died 31/1/05.
8. Mrs G. 58. 18/9/03. 4 months.	None.	Cocain and adrenalin. Whitehead. Left half.	1. Epithelioma.	R.	Good health 1910.
9. Mr M. N. 42. 27/10/03. 2½ years.	Tooth.	Chloroform. Ligature lingual artery and glands. Whitehead Left half.		R.	Not traced.
10. Mrs A. H. 60. 8/1/04. 4 months.	Artificial denture.	Chloroform. Whitehead. Right half.	1. Reported as "granulation" tissue. No evidence of malignancy.	R. R.	Died July 1904. Cervical glands fungating.
11. Mr D. W. 54. 16/2/04. 2 years.		Chloroform. Ligature both lingual arteries, glands, entire tongue and portion of jaw.	1. Epithelioma.	D.	Died pneumonia 4th day.
12. Mr W. J. 52. 9/8/04. 5 months.	Teeth.	Chloroform. Ligature lingual artery and glands. Left half.	1. ,,	R.	Not traced.
13. Mr. J. S. 55. 22/11/04.		Chloroform. Syme. Entire tongue.	1. ,,	R.	Died 18/1/05.

Name, Age, Date, Duration of Disease.	Antecedent Irritation.	Anæsthetic. Operation. Parts Removed.	Microscopic Report on— 1. Tongue. 2. Glands.	Result of Operation.	Further History.
14. Mr J. T. 62. 8/8/04. 14 days (!)		Chloroform. Whitehead. Half tongue and tonsil.	1. Epithelioma.	R.	Died within a year.
15. Mr J. H. 60. 16/11/04. 4 months		Chloroform. Ligature of lingual artery and glands. Whitehead. Left half.	1. ,,	R.	8/2/06. Chloroform. Right half tongue. 10/12/06. Left glands and internal jugular vein. Died, summer 1907.
16. Mr J. F. 63. 1/6/05. 10 weeks.		Chloroform. Ligature lingual artery and removal of glands. Right half tonsilar and palatal region.	1. ,, 2. Glands affected.	R.	Not traced.
17. Mr D. M'L. 53. 25/9/05. 5 months.		Chloroform. Ligature of lingual artery. Whitehead. Left half and portion of lower jaw.	1. Epithelioma.	R.	Not traced.
18. Mr A. W. 59. 17/10/05. 2 months.	Teeth.	Chloroform. Ligature of lingual artery, glands. Right half, pillar of fauces, portion of jaw.	1. ,,	R.	Died 15/6/08.
19. Mr W. D. 50. 9/9/06. 5 weeks.	From pipe.	Chloroform. Ligature of external carotid, glands. Whitehead. Left half.	1. ,, 2. Glands normal.	R.	Not traced.
20. Mr A. F. 64. 28/9/06. 5 months.	Tooth.	Chloroform. Ligature of external carotid, glands. Whitehead. Left half and tonsil.	1. Epithelioma.	R.	Died 1/6/07.
21. Mr J. B. 61. 18/4/07. 4 months.	Teeth.	Chloroform. Ligature of lingual artery, glands. Right half and lower jaw.	1. ,, 2. Glands normal.	R.	Not traced.
22. Mr J. W. 50. 17/4/07. 1 year.	Teeth.	Chloroform. Ligature of lingual artery, glands. Right half tonsil and lower jaw.	1. Epithelioma.	D.	Died 8th day Pneumonia, diarrhœa, suppression of urine.
23. Mr E. B. 54. 9/10/07. 3 months.	Artificial denture.	*a*. Cocain and adrenalin. Half. *b*. Chloroform. Butlin.	1. ,, 2. Glands normal.	R.	Well 1910.
24. Mr A. D. 43. 10/12/07. 3 months.	Teeth.	Chloroform. Ligature of external carotid, glands. Right half, pharyngeal wall. Tracheotomy.	1. Epithelioma.	D.	Died 4th day.
25. Mr T. A. 53. 10/12/07. 3 months.		*a*. Chloroform. Syme. Right half pillar of fauces. *b*. Butlin.	1. ,,	R.	Hale and well 1910.

Name, Age, Date, Duration of Disease.	Antecedent Irritation.	Anæsthetic. Operation. Parts Removed.	Microscopic Report on— 1. Tongue. 2. Glands.	Result of Operation.	Further History.
26. Mr T. F. 57. 21/1/08. 10 months.		*a.* Cocain and adrenalin. Right half. *b.* Chloroform and ether. Butlin.	1. Epithelioma. 2. Glands normal.	R.	Died within a year.
27. Mr A. T. 39. 2/3/08. 3 months.		Chloroform. Syme. Half tonsil, pillar of fauces, pharyngeal wall.	1. Epithelioma.	D.	Shock (?) No section.
28. Mr A. C. 49. 3/4/08. 5 weeks.	Teeth.	*a.* Chloroform. Right half. *b.* Chloroform. Butlin.	1. ,,	R.	Died 14/5/09.
29. Mr J. C. 40. 2/5/08. 9 months.	Teeth.	*a.* Cocain and adrenalin. Right half. *b.* Chloro-ether. Butlin.	1. ,,	R.	17/10/08. Glands removed. 15/6/09. Glands removed. 12/5/10. Looks and speaks well, 3 small painful nodules (?) opposite carotid bifurcation, supra-clavicular region and over sternomastoid.
30. Mr J. B. W. 47. 15/6/08. 5 weeks.	Tooth.	*a.* Cocain and adrenalin. Syme. Entire. *b.* Chloro-ether. Butlin.	1. ,,	D.	Sudden cardiac failure 2nd day. Commencing pneumonia right lung. Excessive fattychanges in organs.
31. Mr J. O. 56. 4/9/08. 2 months.	Teeth.	*a.* Cocain and adrenalin. Right half. *b.* Chloro-ether. Butlin.	1. ,, 2. Glands infected.	R.	Hale and well 1910.
32. Mr A. G. 71. 10/9/08. 6 months.		Chloroform. Entire tongue and mesial portion of jaw.	1. Epithelioma.	D.	Died 5th day. Pneumonia.
33. Mr F. G. 57. 6/1/09. 3 months.	Tooth.	*a.* Cocain and adrenalin. Right half. *b.* Chloro-ether. Butlin.	1. ,, 2. Upper glands infected, mid and lower normal.	R.	Died 14/5/09.

Name, Age, Date, Duration of Disease.	Antecedent Irritation.	Anæsthetic. Operation. Parts Removed.	Microscopic Report on— 1. Tongue. 2. Glands.	Result of Operation.	Further History.
34. Mrs Ross. 60. 17/1/09. 2 months.	Scald of mouth with hot porridge.	*a.* Cocain and adrenalin. Left half. *b.* Chloro-ether. Butlin.	1. Epithelioma. 2. Upper glands infected, mid and lower normal.	R.	April 1910. Seemed well till a week before death, when glands of opposite side ulcerated and fatal hæmorrhage followed.
35. Mr J. W. 70. 13/3/09. 3 months.	Sharp tooth.	Cocain and adrenalin. Right half.	1. Epithelioma.	R.	Died 8/2/10.
36. Mr J. C. 59. 8/6/09. 6 months.	Teeth.	*a.* Cocain and adrenalin. Right half. *b.* Chloro-ether. Butlin.	1. ,, 2. Glands normal.	R.	Well 1910.
37. Mr D. B. 49. 10/10/09. 8 months.	Teeth.	*a.* Chloroform. Right half and jaw. *b.* Chloro-ether. Butlin.	1. Epithelioma. 2. One large gland extensively affected, bad condition capsule, and infected adjacent area. Glands normal in high carotid region and cervic neck.	R.	Well 1910.
38. Mr A. P. 50. 24/7/09. 8 weeks.	Tooth.	*a.* Cocain and adrenalin. Right half. *b.* Chloro-ether. Glands.	1. Epithelioma.	R.	Well 1910.
39. Mr J. T. 47. 16/11/08. 3 months.		*a.* Cocain and adrenalin. Right half. *b.* Chloro-ether. Butlin.	1. ,, 2. Glands infected.	R.	Died 14/7/09, with symptoms of spinal metastasis.
40. Mr W. S. 51. 3/11/09. 12 months.		*a* Cocain and adrenalin. Left half. *b.* Chloro-ether. Butlin.	1. Epithelioma 2. No definite infection, but suspicious area in glands from spinal accessory nerve.	R.	Very well 1910.
41. Mr R. A. 65. 6/2/09. 5 months.	Teeth.	Chloroform. Left half tongue, portion of jaw.	1. Epithelioma.	R.	Not traced.
42. Mr A. F. 60. 5/6/09. 2 months.		*a.* Cocain and adrenalin. Right half. *b.* Chloro-ether. Butlin.	1. ,, 2. Glands normal.	R.	Well 1910.

Name, Age, Date, Duration of Disease.	Antecedent Irritation.	Anæsthetic. Operation. Parts Removed.	Microscopic Report on— 1. Tongue. 2. Glands.	Result of Operation.	Further History.
43. Mr P. C. 64. 12/7/09. 7 months.		*a*. Cocain and adrenalin. Whole tongue *b*. Butlin (Mr Dowden).	1. Epithelioma. 2. Glands invaded right side, left side normal.	R.	Return glands left side of neck, removed 16/12/09.
44. Mr J. M. 73. 13/11/09. 12 months.		Cocain and adrenalin. Left half.	1. Epithelioma.	R.	Not traced.
45. Mr J. H. 70. 18/12/09. 1 month.		Cocain and adrenalin. Right half.	1. ,,	R.	Well 1910.
46. Mr J. G. 57. 21/12/09. 5 weeks.	Tooth.	*a*. Cocain and adrenalin. Right half. *b*. Chloro-ether. Butlin.	1. ,, 2. Highest gland no malignant cells, submaxillary lymphatic gland and that at carotid bifurcation infiltrated.	R.	Well 1910.
47. Mr R. H. 62. 25/1/10. 7 weeks.	Tooth.	*a*. Cocain and adrenalin. Syme. Entire tongue. *b* Butlin.	1. Epithelioma. 2. Higher glands normal, lowest extensively infected.	R.	12/5/10. Local return in tongue and alveolus.

Discussion.

Mr C. W. Cathcart said Professor Caird had done a great service in bringing together his experience, which must have been larger than any of the other surgeons in the Infirmary, and would compare very favourably with the statistics of other surgeons. He had not himself such a large proportion of recoveries. As regards the malignancy of malignant disease there was a great difference; carcinoma was apt to be put down as a malignant disease, and all forms of carcinoma thought to be of the same type; his experience was that there were great varieties of cancer in such a region as the tongue—some cases ran on almost as rapidly as an inflammation, while other cases went on for a considerable time and a successful result was obtained. The adoption of the method of clearing out the glands was of comparatively recent date; personally he had found that a great many of the unfavourable results

were found in those cases in which only a few of the enlarged glands were removed. The local anæsthesia method he had not tried. He thought Butlin's method very valuable, allowing the surgeon to work unimpeded, thereby shortening the period of operation and diminishing the risk of loss of blood. He did not think any one particular method should be adopted; in a comparatively mild case Whitehead's method was quite sufficient; where there was much inflammation of the mouth and tongue then one of the other methods should be adopted: it was unnecessary to adopt the same method in all cases.

Professor Alexis Thomson thanked Professor Caird for his paper, which was given in his usual attractive way. He described this operation for carcinoma of the tongue almost as if it were a pleasure to perform it—give the local anæsthesia one day, the patient helps you through, and some ten days later the gland is removed. He agreed with Mr Cathcart that Professor Caird's results must be better than those of the other surgeons in the Infirmary, probably owing to his great experience and knowledge in dealing with these particular cases.

The weak point in operations on the tongue and lip was that, after taking out the local disease, they missed a couple of inches, and went down to the neck, apparently thinking that there was some immunity or protection in the intervening tissue; it was unlike cancer of the breast, where the operator goes from the breast to the axilla, taking out the vessels as well as the glands. His experience showed that it was necessary to have a continuity down to the neck; he had to record a series of fatal cases where the tongue operation had been done and the glands removed by Butlin's operation. The pathologist said there was no malignant disease and a good prognosis was given, but the patient came back again, and the inference was that the disease was there from the beginning. It required a great deal of courage, both on the part of the surgeon and the patient, in the case of a small epithelioma of the tongue, to decide on having a big operation done; he believed there was no real chance of success otherwise. Most cases do not live more than three years, yet the disease at the beginning is usually very local.

He would like to say a word for the pathologist. A little bit is taken off the top of an ulcer or tumour and sent to the pathologist, who examines it and sends back a report which is not thought

satisfactory; the growth ought to be cocained and a large part taken out and sent to be examined, when no doubt a very different report would be given.

Professor Caird, in replying, said as regards the malignancy of carcinoma, he thought carcinoma of the tongue was most malignant, there was no such thing as a quiescent condition. A patient of his, an old gentleman, aged 60, had a small papilloma of the tongue; it seemed a typical case, was quite localised and was removed, but within a year he died from the disease. With regard to the intervening tissue, after removing half of the tongue and performing Butlin's operation, one would expect to get a recurrence about the floor of the mouth, that however did not occur; if it were generally developed about the floor of the mouth that would be another argument in favour of the more radical operation. The more radical operation would give more room, but the question would be whether the mortality would be as low, or whether other risks were likely to follow.

2. ON RECURRENT ENLARGEMENT OF THE SALIVARY GLANDS.

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The comparative paucity of literature on the subject of recurring enlargement of the salivary glands, is an indication either that the occurrence is somewhat uncommon, or that the condition is one of little importance. A considerable experience of glandular enlargements is likely to be required, before even a few of such cases can be collected in any individual practice, and on this very account the chances of missing the diagnosis are increased. Certain it is that the condition may exist in so slight a degree that no advice is sought, and it may be only when frequent recurrence increases the patient's discomfort or merely stimulates his curiosity, or because an acute exacerbation has supervened, that he submits the condition to professional investigation. Again, so far as the affection of the parotid glands is concerned, the diagnosis of mumps may be made, and unwarrantable isolation enforced, under the belief that the patient is suffering from a contagious disease.

In an experience of some fifteen hundred cases of glandular enlargement I have notes of but five cases of recurrent enlargement of the salivary glands, and of these three were submaxillary and two parotid. It is doubtful if the parotid affection be more common than the submaxillary, though it is more apparent, and therefore more likely to enforce consideration; and I have not been able to find any report in which the condition was recognised in the sublingual glands. My cases are as follows :—

Case I.—A butcher, aged 24, a healthy, well-developed man, came under my observation in 1899 complaining of a recurring swelling under the right jaw, of fully seven weeks' duration. He noticed that, when eating, his neck under the right jaw became swollen. Since its onset the swelling had been constantly present, but only became obvious at meal times, beginning when he commenced to eat, and being largest when he had finished. It had become more distinct during the last two weeks. Between meals there was a slight hardness but no tenderness at the site of swelling. On examination the right submaxillary gland was found to be somewhat swollen and firm. No calculus could be felt, and firm pressure on the gland was slightly painful. He was under my observation for twenty days, during which time the condition remained unchanged. He then had belladonna ointment applied by inunction over the tumour, and, whether by coincidence or otherwise, in a day or two the trouble had ceased. I did not see him again for ten years, until the 10th of July 1909, when the condition was once more present. He informed me that he had had an annual recurrence, generally in the springtime, but it gave him no special concern. The obvious swelling could now be easily palpated bimanually with one finger in the mouth. It was firm and slightly painful on pressure, and eating so distinctly increased his discomfort that at times he preferred not to eat. Within a few days it again disappeared.

Case II.—A weaver, aged 23, complained of recurrent swelling under the right jaw, of one year's duration. This patient had a small sebaceous cyst in the skin at the angle of the jaw, and immediately in front of this a recurrent swelling would appear during eating, or if she talked a great deal. It formed a rounded tumour about the size of a marble, evidently submaxillary, and contained

no calculus. On each occasion all discomfort vanished within two or three minutes after she had finished eating.

Case III.—A married woman, aged 44, presented a recurrent swelling of the right submaxillary gland which had existed off and on for two months. She thought she had caught cold shortly before its appearance, for she had had a sore throat and a cold in the head, and it was then that the right submaxillary gland first swelled. On palpation in the mouth the enlarged gland could be distinctly felt, with an abnormal hardness in consistency.

These cases all relate to submaxillary affections. The following are two in which the parotid participated :—

Case IV.—An unmarried lady, aged 35, consulted me in September 1907 for a swelling of the right parotid which had recurred during eighteen months. Her first attack occurred in February 1906. It was ushered in somewhat suddenly at night, with pain about the upper part of the gland, and by the following morning the entire gland was enlarged. The doctor whom she consulted pronounced it " mumps," and she was isolated for twenty-four days. There were no other cases of mumps in the neighbourhood, nor did the children in the house develop mumps. Fomentations were applied and the parotid region painted with iodine. The swelling remained through fully a fortnight, and she could not open her mouth during the first day or two. There was some pyrexia during the first ten days. The temperature never rose to over 100°, but did not come to normal while the swelling lasted. Her second attack came on in April 1907. It came on suddenly during the night, and when she wakened with the pain she found the parotid swollen about the upper part or socia. She was a week indoors. The temperature again continued raised, but only slightly, and the doctor kept her in bed on that account. Pain only existed during the first day. Her third attack came on with some tenderness in September 1908 during the night, and the discomfort continuing, she found next morning that the gland was swollen and she could not open her mouth easily nor fully rotate her head. The swelling was hard, situated under the jaw, and behind the ascending ramus. She remained in bed, and in the afternoon took her temperature and found it to be 99.8°. The pain was as usual worse during and after eating. The swelling

was distinctly less on the second day, and the lower part of the gland formed a vaguely defined enlargement over and behind the body of the right lower jaw, and was slightly tender to touch. On this, as on the first occasion, a cold in the head preceded the attack. She is very liable to "little blisters" in the mouth, though all her teeth are artificial. I only had the opportunity of observing one other attack before the patient left the district, though she had had a fourth attack in January in which the same gland was involved, the affection lasting six days. In February 1908 she wakened with a headache and had pain about the socia parotidis on the left side. She felt miserable all evening, and went to bed shivery and out of sorts. Next day there was evidence of a little hardness at the lower part of the gland at the commencement of the duct. This disappeared in a day or two. Two years elapsed before she had another attack. On the morning of 20th January 1910 she awoke feeling the cheek swollen and stiff. It was very painful all day, and her temperature remained at 99.8°. She fomented it. Next morning the pain was less though the swelling was more distinct, but they gradually subsided, until in a day or two the gland was again normal.

This case constitutes the type of the recurrent enlargement of the parotid, and is characterised by absence of cause, and by the constitutional disturbance and local manifestations. In the following case there is present the possibility of some occult relationship between the local condition and the general waxy disease from which the patient suffered, though the constitutional disturbance due to the parotitis has not latterly been recognisable :—

Case V.—A girl, aged 19, by occupation a machinist, was the subject of tuberculous disease of the hip with formation of abscesses, and subsequently sinuses. This had persisted in spite of treatment. There had been repeated scrapings ; the head of the femur had been excised in 1908, and disarticulation at the hip performed a year later. She had developed during the past three years extensive waxy disease of the viscera, with diarrhœa, albuminuria, ascites, and general anasarca.

In January 1909 the right parotid gland swelled for the first time. It came on during eating, and the swelling was pretty considerable, and interfered with her opening her mouth. No note of it was made at the time, though she was then in hospital,

and the irregularity of her temperature chart cannot be said to have indicated a feverishness which might not have been sufficiently explained by her tuberculous condition. The swelling recurred frequently thereafter in the right gland, and three months later the left became affected, but never to the same extent, or with as much discomfort as the right. To begin with, the swelling of either gland did not come on invariably during mastication, but latterly was much less frequently absent. It never appeared when she was not eating, but should she feel sick and about to vomit, the swelling of one or both parotids accompanied the pre-emetic salivary flow, and was always exceptionally well marked under these conditions. At other times the swelling only came when she chewed, and especially if she ate fruit. The onset varied in earliness, but a few minutes after she ceased to eat the swelling had entirely gone. She was a somewhat pasty-faced girl of the usual type where tuberculous disease is in combination with a general waxy affection, but bright and cheerful and decidedly intelligent. There was never anything abnormal to be seen in the mouth, nor, during the swelling, could any parotid secretion be expressed from the ducts. The swelling still gave her the square-faced appearance of mumps, but apparently was accompanied by little discomfort.

From what literature I have been able to gather on the subject the parotid affection would appear to be the more common, though in my limited experience it is the reverse. Perhaps the greater size and importance of the parotid, and its greater fixity in a tenser sheath, may account for more frequent reports of its affection being recorded than of the submaxillary, where considerable swelling causes little inconvenience, and is much more likely to escape observation. Another difference between these two affections seems to be that, though the submaxillary remains single, the parotid affection tends ultimately to become bilateral. The physiological, and especially the anatomical, differences between the parotid and submaxillary glands are no mean factors in the symptomatology.

Predisposing causes are not always to be found. In none of my patients could such be certainly ascertained. One had a sore throat and cold in the head preceding her submaxillary attack, and in my first case of parotid affection the lady was the subject of constipation, and was liable to "little blisters in the mouth."

In the second of my parotid cases the commonness of waxy affection in association with wasting diseases accompanied by suppuration, and the rarity of recurrent salivary enlargement, enables one to disassociate the two conditions. Waxy degeneration does not affect epithelium, and therefore cannot be the sole reason of the salivary affection.

Dyspepsia and constipation, as mentioned by Cocking (1), may be a predisposing combination, because furring of the tongue and various mouth troubles (such as aphthous patches, stomatitis, etc.), are likely accompaniments. A special predisposing cause seems to exist in submaxillary lithiasis. Multiple passage of calculi is uncommon, though in one case (a man aged 39) no less than four recurrences were noted (14), but it is still more rare that the extrusion of a calculus is followed by *recurrent* enlargement of the gland. Hakes (5), however, records that such occurred after the escape of a second submaxillary calculus, where apparently some subsequent cicatricial contraction had narrowed the orifice of the duct. A curious case of toleration of a submaxillary calculus during five years, in a man aged 20, and causing during that time a recurrent submaxillary enlargement, is recorded by Pravaz (19), but the case is reported too soon after extrusion of the calculus to make sure that subsequently there was no further recurrence.

No writer suggests, as a predisposing cause, any inflammation of the submaxillary which could bear the same relation to it, as is claimed for mumps in the parotid affection, though the submaxillary and sublingual glands may or may not be implicated in mumps (16). In the light of subsequent development one must often hesitate to admit the initial affection as true "mumps." The literature of the subject teems with such errors as regards the parotid, and my fourth case shows how readily such a mistake might be made. There is something more wanted, however, than mere local symptoms with a constitutional disturbance to prove the existence of mumps. It is desirable that a possibility of infection be elicited, or at least the existence of mumps in the neighbourhood.

Permanent enlargement of the parotids after mumps must, if it exist at all, be of the greatest rarity. Dr John Thomson (6) exhibited, and has published (17), a case of chronic enlargement of both parotids in a mentally defective lad of 15 years. It had existed since "an ordinary attack of mumps" seven years previously, other members of the family having had the disease at

the same time. An isolated coincidence makes no law. Without mumps, however, ill-health and poor development are occasionally accompanied by an apparently symptomless enlargement of the parotids. Johnson (2) reports one example of this in a thin, anæmic girl aged 13, and another in an ill-looked-after and ill-nourished infant of fifteen months, but, as in Thomson's case, though the *enlargement* was present, there was no *recurrence*. It seems straining probabilities to record as the exciting cause "mumps" ten years and thirty years before the onset of the recurrent affection (Hutchinson (8)). At the same time, it is not possible to disassociate the inflammation of mumps from the affection of the parotid. Bouchut (15) is reported to have stated in a communication on the pathology of mumps in the Academy of Science that "mumps," according to his researches, really arise from retention of saliva caused by catarrhal inflammation of the parotid duct. Under the influence of this catarrh of the excretory canal a temporary obstruction is caused, which retains the saliva. In children seized with this affection while in good health the disease is not a serious one, he says, for suppuration does not take place. Though this theory will not hold good, and he is wrong in supposing that suppuration in mumps never takes place in strong children, and he seems to ignore the infective nature of the disease, it is interesting as bearing on one cause of parotid enlargement.

That mumps should be succeeded by permanent enlargement of the parotid glands is certainly more intelligible than that the enlargement should be recurrent. The fact remains, nevertheless, that many of those cases of recurrent swelling are said to have been preceded by an attack of mumps. One can call to mind in this respect many cases of glandular enlargement which ultimately proved to be tuberculous, in which the initial swelling was originally pronounced to be mumps. Before admitting that the original affection in recurrent swelling of the parotids was mumps, one requires some evidence of contagion, for probably that is the most, if not the only, characteristic attribute of mumps. The swelling, the pain and tenderness, and the alteration of the secretion, are common to both mumps and recurrent parotitis, but the interference with hearing is less common in the latter, and the fever at the commencement of mumps is apt to be higher, and it is to be noted that in mumps both glands are far more commonly affected, and the second one at a much shorter interval after the first, than in recurrent parotid enlargement.

One cannot pass from this part of the subject without reference to the condition which goes by the name of "Mikulicz's disease." This "disease" was originally described by Mikulicz in relation to chronic enlargement of the lachrymal glands, and it was subsequently found by others that the affection later involves the salivary glands, and in many cases the order of incidence is reversed. Campbell's (20) admirable essay on the subject is most instructive, and his *résumé* of fifty-five collected cases interesting. There is a smooth, painless, gradual enlargement of the affected glands, with no fever or malaise, but sometimes with phenomena due to lack of glandular secretion. The relationship which Campbell claims to exist between Mikulicz's disease and leukæmia and pseudoleukæmia has no bearing on the subject of this paper further than that in neither, nor in Mikulicz's disease, is there any note of *recurrence* being a feature, or even present, in the course of the affection. There may be a similarity in this, that in Mikulicz's disease the origin is presumably microbic, and its initiation often preceded by some "pre-existing lesion of the mucous membrane" (20).

The exciting cause of the enlargement is almost invariably the act of eating. It is natural that with increased secretion and flow of saliva towards the mouth, any localised obstruction by narrowing of the duct, either as its ostium or in its canal, should manifest itself by damming back the secretion and producing consequent enlargement and pain. In my fifth case it is to be remarked that the swelling varied in rapidity and severity with the quality of the food taken, the "obstruction" manifesting itself in proportion to the copiousness and rapidity of the salivary secretion. This patient specified apples as being peculiarly liable to produce the swelling, just as in Claisse's (18) case "any acid substance, particularly oranges," acted as a special stimulant. A difficulty in the passage of saliva would be more likely to occur were the secretion from any cause more thick or viscid than natural. With the cessation of eating comes diminished secretion, and the pain is relieved at once and the swelling soon after, unless the obtruction be complete. It is not likely that if the duct were at all permeable the damming back would cause permanent enlargement, but frequent recurrences, or long duration of the enlargement, might in course of time produce permanent alterations by interacinous hyperplasia and dilatation of ducts. An unusual case is recorded by Jonathan Hutchinson (8) (Case II.), in which the swelling did

not go down " until the patient (a woman of 54) took food." This seems to indicate a loss of tone and power of expulsive effort which the stimulation of eating temporarily overcame, and so assisted evacuation.

In the affection of the submaxillary the onset appears to be more rapid than in the case of the parotid, though the onset never merits the term " sudden." It is not uncommon that the first attack is more severe than the subsequent ones, but it is not recorded *in extenso*. The very rapidity of onset may suggest a calculus, and it is possible that such affection of the glands may remain unpublished, under the impression that a calculus has been the cause and has been missed. Hutchinson (7) records such a case. The recurrence of the attacks, however, ought to raise doubts as to the correctness of the original diagnosis, and it is to be remembered that the calculus may be small and overlooked, as in Pravaz's (19) case. Nor is the difficulty of excluding calculus lessened by the fact that the passage of a calculus, with injury to the duct, may occasionally be the primary cause of subsequent recurrent swelling (5).

Very much allied to this in its mechanism is the obstruction caused by the alteration of the secretion, the condition known as *sialodochitis fibrinosa*. The duct becomes blocked with muco-purulent secretion, and, on this being squeezed out, a flow of " fluid," " secretion," " sour water " takes place into the mouth and the swelling subsides. One cannot accept this alteration in the secretion without inquiring into its cause.

Quite recently a case, apparently of this nature, was referred to me by Dr J. Mackie Whyte. The patient, a married lady, aged 33, was the subject of recurrent swelling of the left parotid gland during twelve years, though always of a mild type. Her family history is good, and her personal history unexceptionable. She had had no previous illness, had borne two children, and had no history of gastro-intestinal derangements. She remembered the initial attack well, because she was preparing to go to a party when, " in a minute," the swelling came on. It was not accompanied by any pain, nor malaise, nor feverishness. It seems to have reached its maximum rapidly, and then during the following three days gradually subsided. It recurred thereafter at intervals of three to eight weeks. The attacks had no relation to menstruation, and she could never attribute any cause for an onset. Each

attack had been much the same as, but never worse than, the first. Sometimes she feels swelling over the malar rather than the parotid itself, and this I can confirm from my own observation, and sometimes she feels the swelling over the horizontal ramus in front of the body of the lower jaw. At times she feels her face "like a drum, tense and strained, around and behind the ear," and when this has lasted some time a gush of saliva "with a slightly sour taste" takes place into the mouth and immediately she gets relief. She has seen the saliva rush into the mouth, and, so far as her observation goes, it is normal in appearance and consistency. She is never able to produce this flow by pressure or suction. It is to be observed that there are here, perhaps, two conditions, the former being characterised by a recurrent swelling of the face in parts not anatomically closely corresponding with the area occupied by the parotid gland, the latter being characterised by an undoubted involvement of the gland and a retention of secretion. There is another point of great interest. The former variety of swelling seldom comes on during the day, but is generally there when she rises in the morning. It gives her no discomfort, and she does not know it is there until she looks in the glass, when she can verify the observation by touch, and she says the skin looks shiny and feels slightly hot, and there is never a subjective escape of saliva during its existence nor with its subsidence. The latter variety comes on much more frequently during eating than at any other time, is characterised by more discomfort, and disappears with a salivary flow. Its onset, too, is very frequently accompanied by a most uncomfortable itchy feeling at a localised spot rather more than an inch in front of the lower part of the external auditory meatus; in other words, at the commencement of the parotid duct. Her teeth are good. The papilla and the submucous course of the duct are more pronounced than on the opposite side. Six years ago she was for a time under the care of Mr J. H. Nicoll of Glasgow who probed the duct and did not find any calculus. He considered the case to be one of angioneurotic œdema, and I believe this to be the explanation of that variety of swelling to which she is liable, which does not correspond with the parotid gland, gives no discomfort, and is not followed by a salivary flow; but to explain the syndrome which constitutes the other variety of swelling, we are compelled to consider it a sialodochitis fibrinosa. It differs from true recurrent parotitis, in that its original onset was extremely sudden, the swelling has never been accompanied by any

constitutional disturbance, and its subsidence is synchronous with a sudden copious flow of saliva from the duct.

Morestin (3), in Dentu and Delbet's *Surgery*, contributes a special paragraph on "Inflammations limitées aux conduits excréteurs des glandes salivaires," naming each "stenonitis" and "whartonitis." Of the former he refers to a case recorded by Demarquay in 1856, where the parotid duct was blocked with pus and distended with secretion and gas, and two cases under Chassaignac which simulated parotitis, but the inflammation and suppuration were limited to the excretory ducts. He mentions an observation of Terrier's where retention was caused by an aphthous patch at the opening of the parotid duct. The submaxillary condition he ascribes to any lesion at the ostium of the duct, and says that a simple whartonitis may result in the formation of a plug of epithelial cells and mucus which may produce temporary obstruction, and the escape of which is quickly followed by relief of the symptoms. Digital pressure on the gland or even strong suction by the mouth (19) may be sufficient to produce the desired clearing of the duct. Similarly there may be alteration in the secretion escaping from the parotid duct. Johnson's (2) patient had ropy mucus which could be squeezed out by pressure. He considers that the swelling is caused by "interference with the escape of the gland's secretion due to the inflammation of the lining of Stenson's duct." The socia parotidis alone may be affected, because it has a small duct of its own entering the main duct. In Kussmaul's case a traumatism was suggested as the cause, but the later bilateral affection made this hypothesis untenable.

Rise of temperature at the commencement of the attack seems to be a feature in the typical parotid affection. This it has in common with mumps, but the feverishness is rarely so high in the former, whereas relapses, though known in the latter, do not continue through a succession of years, as recurrent swelling of the parotid may. It is not usual to find the temperature rise to 103.6° F. as in Johnson's (2) case, but rather to find, as recorded by Hutchinson (8) (Case I.), that the initial onset is accompanied by "a sharp feverish attack," while subsequent recurrences are preceded by malaise and "aching all over," and this coincides with my observation. Kussmaul (11) states that in his patient there never was fever, but it is not clear that his observation exhausts the details of the initial affection.

Useful generalisation can scarcely be made from the small

number of cases recorded. Out of twenty-two, including my own, the parotid was affected in equal frequency with the submaxillary. Of the former, eight were females and three were males, and of the submaxillary affection, seven were males and two were females, while in two cases the sex was not specified in the reports. When the age has been stated, the parotid has been affected between the age of 26 and 54 (four of these were below 30, three below 40, one below 50, and one below 60). In Johnson's (2) (Case I.), a female, aged 13, had one parotid affected. In this case the rapid onset with feverishness, the duration, and absence of suppuration make it probable that it was the first appearance of what would afterwards become a recurrent parotitis. In the submaxillary affection, if we exclude a child of 9 years (5), where the condition followed the passage of calculi, and Pravaz's (19) case, aged 15, also due to the presence of a calculus, the ages given are 23, 24, 30, 40, 44, and there are other four recorded as "adults" merely, the ages not being stated. The conditions, apart from an occasional accidental occurrence, such as a calculus in youth, would seem to be one of adult life. Indeed it is in adult life, when nervous affections are common, when secretions are apt to undergo alteration, when there may be dyspepsia, stomatitis, and carious teeth, and when the tissues are becoming less resilient, that one would expect to find conditions favourable to the development of recurrent swelling of the salivary glands.

It is natural that an affection of the salivary glands should produce symptoms due to interference with their secretion. Intermittent ptyalism is of very rare occurrence, and probably associated with some nervous disturbance, as in a case recorded (13), which existed for five years in a female melancholic lunatic, aged 38. The other extreme is not nearly so uncommon, where the mouth is affected by extraordinary dryness. On this condition the name "xerostomia" has been bestowed. The dryness may be the only symptom complained of, and there is evidence that its causation may at any rate sometimes be central. The milder forms of mouth dryness are not at all uncommon, and as they are usually accompanied by abnormal sensations in and about the tongue and fauces, such patients comes for advice on account of their fear of cancer. I do not have notes of such a case under 45 years of age; generally they are much older and edentulous, and the duration alone of the symptoms at once leads one to doubt the possibility of malignancy, even before examination.

The tongue is clean, but redder and drier than normal, and a history of variation in the severity of the symptoms is generally elicited.

It seems that prolonged xerostomia is accompanied sooner or later by enlargement of the salivary glands. Thus Battle (4) records the case of a woman, aged 54, whose xerostomia had existed three years before enlargement of the parotids appeared. In Fraser's (9) case all the salivary glands were affected (as well as the lachrymal and sweat glands), but only the parotids are referred to as undergoing enlargement, and that only slightly, though the xerostomia was very severe. In Chappell's (10) first case xerostomia had existed for four years before the parotids enlarged, and enlargement of the submaxillary glands followed later, while subsequent death from paralysis and coma suggested a central lesion ; but in his second case, a woman of 60, with xerostomia, there was no enlargement of any gland. *Per contra*, an affection of the salivary glands or parotids alone for a sufficiently long time would likely result in xerostomia. Hutchinson (8) records the case of a lady, aged 52, whose swelling of the parotids had recurred during twenty years, and the enlargement only became permanent after the supervening xerostomia had existed for four years. In Fraser's (9) case of xerostomia both parotids were swollen, though unequally. In such cases, as there is an absence of secretion in the parotids (and it may be in other secreting glands), the enlargement cannot be looked upon as due to retention. Fraser's patient had several attacks of temporary enlargement, and though there was evident absence of secretion, it is extremely interesting to note that " on one occasion, eight hours after one-sixth of a grain of pilocarpin had been injected subcutaneously, both parotids become enlarged, painful and tender, and continued so for five days, and during the first forty-eight hours of the parotitis the general temperature was pyretic "—an exact counterpart of the initial attack in recurrent parotitis. Fraser concludes that the glands and ducts are not " permanently or importantly injured." To indicate the extensive nature of the affection, Fraser proposes the name " stomo-rhino-ophthalmo-xerosis." In Chappell's (10) case of somewhat similar disease both parotids " would suddenly enlarge at times, and after two or three days gradually subside." This apparently resulted in a permanent enlargement, and the submaxillary glands became similarly affected. In Battle's (4) case, also, both parotids were symmetrically affected.

In these cases the xerostomia, not the swelling, was the chief symptom, though the recurrent enlargement was undoubtedly a feature ; and indeed it appears that in unilateral affection of either gland, xerostomia is not complained of. The other parotid rarely escapes the inflammation in mumps, yet dryness of the mouth is not a common feature, though it may exist, and hypersecretion even is known to have been, though rarely, present (16). The inflammation alone would not account for the xerostomia. In one case the xerostomia (Hutchinson (8)) was worst during exercise, an unusual occurrence, which I suggest may possibly be associated with the drying of the mouth by increased rapidity of breathing. It cannot be denied that a central origin might account for this, and also explain another case of Hutchinson's (8) in which the recurrence was liable to be brought on by fatigue. Perhaps a similar association might be evidenced where the attacks were accompanied with irritability of the bladder, polyuria, and frequency of micturition. It is interesting to note that one of Chappell's cases (10) was associated, when severe, with increased micturition.

Reference has been made above to the tendency unilateral recurrent parotitis has to become bilaterial, but I have been able to find no record of bilateral submaxillary affection. Another point of difference between the affection of these glands is that, unless the submaxillary affection be extraordinarily acute, there is no constitutional disturbance ; whereas in the parotid affection constitutional disturbance is the rule, at least in the initial attack. The duration of each attack is very variable—from a few hours or less to a week or two or even longer.

The interest of the condition does not lie in the treatment, for that appears to be a matter of little moment. Should the attack be exceedingly acute, incision may be necessary, and the absence of specific inflammatory affection seems to make recovery strikingly rapid and complete. Incision was obviously called for in Demarquay's case, where the duct was distended with purulent secretion and gas. In one case Hutchinson (7) adopted this treatment under the belief that the retention was due to a calculus, and he writes that he had never had a case of inflammatory enlargement in connection with the submaxillary where recovery was so rapid, complete, and permanent. He recommends probing the duct, which, if obstruction existed, would be sure to prove beneficial, as it would ensure the evacuation of the contents of the passages more efficiently than compression or suction, though

less so than incision. In acute cases Morestin (3) and others recommend catheterism of the duct, and in chronic cases the injection of iodine solution along the canal. It would seem reasonable that the avoidance of anything which would stimulate, and the exhibition of internal drugs or local applications likely to diminish the gland's secretion, might exert a beneficial influence ; but certainly in none of my cases was surgical interference ever indicated. Indeed the indications for surgical treatment are obscured by the confusion which exists as to the varieties of salivary glandular swellings.

In conclusion it may be stated that intermittent swelling of the salivary glands is due to—(*a*) Causes connected with perverted secretion : (1) sialolithiasis ; (2) sialodochitis fibrinosa ; (3) primary xerostomia. (*b*) Causes connected with the ducts : definite narrowing of the ostium due to (1) trauma (*e.g.* calculus) ; (2) ulceration (*e.g.* stomatitis). (*c*) Causes other than the above, apparently in connection with the glandular function but not yet pathologically defined.

It is under the last category that my cases must come, but it is a lame conclusion which submits that the condition is unexplained. I have, however, no explanation to offer. The contribution is from a clinical standpoint only, for the cases are few and far between, and do not lend themselves to pathological or chemical investigation.

Discussion

Mr Dowden mentioned an instance where he had known two attacks of enlargement of the salivary glands in a friend of his own, he himself having had the opportunity of seeing the second one ; one day when blowing a bugle, an enormous swelling suddenly developed at the side of the patient's face ; it took a few days to go down and was attended with pain and some temperature. On another occasion the exertion of blowing a whistle was followed by a repetition of the sudden swelling of the face.

The *President* said they would agree with him that they were indebted to Mr Greig for bringing this subject before them ; these cases were not common but were most interesting. Personally he remembered having seen in the course of his life some cases of this kind, in particular one very striking case which occurred in the course of cirrhotic Bright's disease, in which very acute recurrent

swelling of both parotids occurred, giving rise to considerable difficulty and then subsiding; the condition occurred more than once in that case. Within the last few months he had seen a gentleman who had a chronic recurrent parotid swelling for which there was no obvious cause. This patient had been seen by one or two London surgeons and also by surgeons in Edinburgh; his ducts had been explored, but no calculus or other condition had been found. Other similar cases had come under his notice, but his observations were passing and trivial and he could not pretend to throw any light on this interesting subject.

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3. FUNGUS INFECTIONS OF THE FINGER-NAILS[1]

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In his handbook of *Diseases of the Skin* (1907), Whitfield states that fungus infections of the nails are probably commoner than is generally supposed. That statement is borne out by the fact that within the last three years I have had the opportunity of observing no fewer than 19 cases; 17 of these were seen as out-patients in the Skin Department of the Royal Infirmary under Dr Norman Walker's care, and to him I am indebted for permission to record them. For one case, which was an out-patient at Leith Hospital, I am indebted to Dr Langwill.

I do not propose to describe each case separately. They fall into three groups, viz.—(1) Ringworm of the nails; (2) Favus of the nails; (3) a Fungus Infection of the nails of unknown origin.

As the cases of ringworm form the most important group, they will be dealt with first. There were 16 of these ringworm cases, 14 of which belonged to Edinburgh and 2 to Leith.

A curious fact was the predominance in the female sex—only 2 being in males. This predominance is difficult to explain. Török says that tinea unguium is commoner in those who manicure the nails, and probably injure them in so doing, but the majority of our patients were not of the class which bestows much care on the nails. It is possible that the frequent washing of infected material, *e.g.* clothes, etc., may be the reason why females so frequently suffer.

Ringworm of the nails may occur at any period of life. The youngest of my cases was 11, and the oldest 58 years of age. The majority were girls of between 20 and 30 years. This age-incidence corresponds more nearly to that of tinea corporis than of tinea capitis.

Ringworm of the nails is very rare in children with ringworm of the scalp. Sabouraud, in l'Ecole Lailler, where there were always about 300 children with ringworm of the scalp, in four years saw only 2 cases where the nails were affected. This infrequency of nail infection in children with ringworm may be partly due to the fact that ringworm of the nails is always due to the

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large-spore fungus, but even amongst children suffering from large-spore tinea of the scalp nail affections are very rare. I think the most probable explanation is, that because tinea capitis so rarely causes any itching there is no tendency to scratch and so infect the nails. The inflamed ringworms (kerion) are also too tender to allow of scratching.

Source of Infection.—The source of infection was difficult to trace in many of the cases. In only 1 of the 16 cases did the patient suggest that the disease was due to an infection from anyone else. The patient blamed the washing of clothes worn by her husband and son, who had what was apparently tinea barbæ. In all the other cases the patient had no idea of the condition being a contagious one. In another case there is also little doubt that the disease was contracted from her brother who, I know, had tinea barbæ at the time when her nails became infected, but as cultures were not made from the brother's beard I cannot say whether or not both conditions were due to the same fungus.

Two of the cases were husband and wife and other two were sisters, and in these cases there was probably either a common source of infection or infection the one from the other.

One woman was undoubtedly infected from her children, who had tinea capitis of large-spore variety. In washing and rubbing their heads she contracted ringworm of the hands and fingers, the disease later spreading to the nails. In this case cultures showed that the same fungus was present in the nails as on the children's heads. This method of infection has been previously described by Vidal, who noticed that nurses who epilated ringworm cases at the Hôpital St Louis in Paris frequently became infected with ringworm of the nails.

In all the other cases absolutely no source of infection could be traced either from previous lesions on their own skins, from infection from their relatives or friends, or from animals. Each patient was particularly questioned with regard to these points.

In two cases there was a history of injury to the nail shortly before the disease began, and it is possible that infection may have taken place at that time.

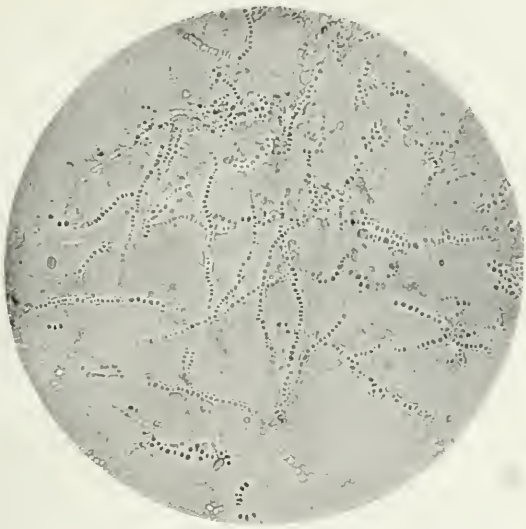
One case was associated with spoon-nails (coilonychia). The spoon-nail was well marked in all the nails, and possibly such nails are more liable to fungus infections than normal ones.

Ringworm of the Finger-nails.

PLATE I.—TO ILLUSTRATE DR R. CRANSTON LOW'S ARTICLE.

Ringworm of the Finger-nails.

PLATE II.—TO ILLUSTRATE DR R. CRANSTON LOW'S ARTICLE.



Ringworm fungus in finger-nail. Mounted in liquor potassæ.
(× 400.)

Same as above. (Oil immersion.)

PLATE III.—TO ILLUSTRATE DR R. CRANSTON LOW'S ARTICLE.

Cultures (about six weeks old on Sabouraud's medium) of fungi from finger-nails.

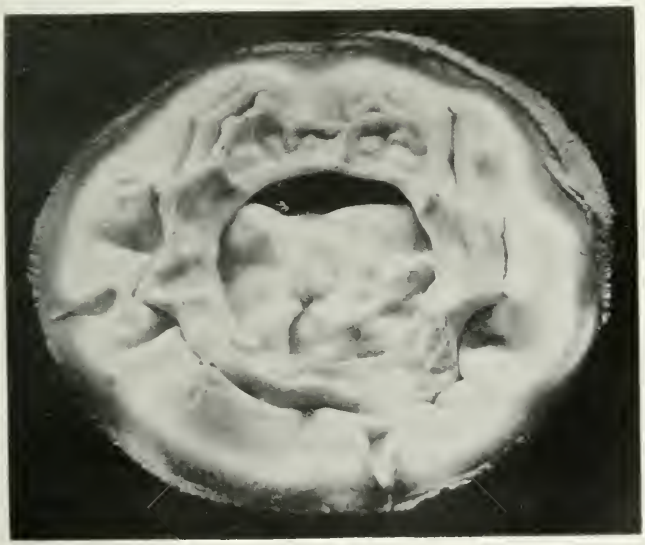
Trichophyton accuminatum.

Trichophyton rosaceum.

Trichophyton crateriforme flavum.

PLATE IV.—TO ILLUSTRATE DR R. CRANSTON LOW'S ARTICLE.

Trichophyton crateriforme flavum.



Pleomorphic form of trichophyton crateriforme flavum.

PLATE V.—TO ILLUSTRATE DR R. CRANSTON LOW'S ARTICLE.

Fungus (unknown variety) in finger-nail.

Fungus from finger-nail (variety unknown). Grown for six weeks on Sabouraud's medium.

PLATE VI.—TO ILLUSTRATE DR R. CRANSTON LOW'S ARTICLE.

Duration.—The duration of the disease varied from three weeks up to seven years, the average time being about four years. Only a very few of the cases had been previously treated, and in some the disease was discovered accidentally, when the patient came complaining of some other condition.

Extent.—The number of nails affected in each case varied from one up to seven. No one nail was more often affected than another. In one-half of the cases both hands were affected, in the other half only one hand. In the 8 cases of the latter group, 4 showed the disease on the right hand and 4 on the left. In none of the cases were the toe-nails affected.

Diagnosis.—The diagnosis in all cases was comparatively easily made, the appearance of the nails being very constant. In all previously recorded cases the authors lay stress on the splitting up and splintering off of the free margin of the nail. This is characteristic of the disease, but as the patients often clip away any loose parts, the nail frequently does not show this to any great degree. The colour of the nails has been variously described from a yellow-white to a brown or black colour. In nearly all my cases I have found that there are definite zones presenting different colours and appearances, these zones being very constant in arrangement. The infection seems to take place from the under surface of the free margin of the nail. At the tip the nail becomes undermined, dark in colour, and softer than normal, so that if a blunt instrument be inserted under it some of the softened nail substance can be easily removed. The disease slowly spreads up the nail, and as it spreads the nail becomes more broken up, till finally, when the whole of it is infected, the free margin breaks off in splinters of a dirty white colour. These broken parts at the tip are easily removed. Then comes a zone where the nail is light yellow in colour, longitudinally striped with darker lines. At this level pieces of the nail can be scraped off only with difficulty. Further up still there is a peculiar dark yellowish-red zone, fairly smooth on the surface, and showing very little tendency to split up. These three zones were distinctly seen in most of the affected nails, and appear to me to be very characteristic of the condition, more especially the yellow-red zone at the upper part. This yellow-red smooth area represents the spreading edge, where the infection is only just beginning. In the light yellow area further down, the nail is permeated with the fungus, but not yet sufficiently so as to cause the nail to break up. The tip is the part of the nail which

has been longest infected, is thoroughly permeated by the fungus, and so is split up and cast off. These zones look as if they were stationary, whereas in reality the upward growth of the fungus is simply keeping pace with the downward growth of the nail.

In addition to these colour changes and splitting up, there is also in many cases a heaping up of the nail into irregularly arranged prominences with dark blackish-brown depressions between. When the nail remains flat the nail substance is probably the only part affected with the fungus, but when it becomes raised up and distorted the nail-bed is probably also affected. In many cases the lunule has disappeared altogether, and the tissues at the base of the nail are red and somewhat swollen. In such cases the nail is very distorted, and it is an indication probably that the growing nail matrix is invaded by the fungus. Apart from the disfigurement there are no symptoms associated with the disease.

Microscopic Appearances.—In all the cases the diagnosis was confirmed by microscopic examination. Pieces of the nail were scraped off with a sharp spoon or clipped off with scissors, put in liquor potassæ (6 per cent.) and gently heated for a few minutes. They all showed the same appearances. Inside the nail substance branching mycelium and spores are to be seen. The spores are square in shape and arranged very characteristically in rows. If the fungus is not visible at once the pieces of nail should be left in the caustic potash over night, and if it is present at all there is no difficulty in seeing it next morning. In some of the cases fungus was not found in the first pieces of nail examined. Therefore in all cases of suspected fungus disease of the nails scrapings should be repeatedly examined. I would also strongly recommend that in every case where a patient presents a splitting up of the nails, which does not affect all of them, that a thorough examination be made for the presence of fungus. Finding of the typical fungus is proof positive of ringworm, but the failure to do so on first examination does not necessarily exclude that disease.

An attempt was made to stain the fungus in the nails by gentian violet and other stains, but the results were not satisfactory. For all practical purposes the caustic potash method is quite sufficient.

Cultures.—In order to determine the kind of fungus present, cultures were made from the nails of 8 of the patients. Pieces of nail were scraped or clipped off and placed for 2 or 3 minutes in

absolute alcohol. Whilst in the alcohol they were broken up into as small pieces as possible. These were allowed to dry for a few minutes and then implanted on Sabouraud's proof medium, which contains 4 per cent. maltose, 10 per cent. peptone, and 1.5 per cent. agar. The cultures were kept at a temperature of 20° C. Some of them showed a pure culture of the fungus, but the majority contained also staphylococci, various yeasts, and penicillium glaucum, but by early subculture the fungus was easily isolated. In 5 of the cases trichophyton crateriforme flavum (primrose coloured) was obtained. These cultures are the same as that described by Colcott Fox in cases of ringworm of the body and scalp in London. This culture begins as a white downy growth, which very soon shows a central depression and the characteristic sulphur yellow colour. The central depression enlarges till a typical crater is formed. In some there are radiating depressions running outwards from the crater in the centre, in others these are not marked. The surface of the culture looks powdery, and in old cultures the centre crater area becomes brownish in colour and cracked. If these cultures are kept for some time a change takes place. There appears a whitish growth, which gradually spreads over the yellow culture and obscures it. This is the pleomorphic change which occurs in all old ringworm cultures which are grown on media containing sugar, such as maltose. Subcultures from this altered culture give very beautiful whitish-yellow growths which are distinctly crateriform.

The cultures from other two of my cases correspond to the trichophyton rosaceum, which is the large-spored fungus found on birds. The young cultures are fluffy and white, but soon show a delicate pink colour, and in some of the growths a yellowish tinge also in the centre. They are raised in the centre and have a few radiating depressions. These cultures also rapidly tend to show a pure white pleomorphic growth.

From another case peculiar cultures were obtained. At first they were contaminated by staphylococcus citreus. They were repeatedly subcultured and a growth obtained which had a pink tinge in places. On making subculture from the white and pink parts two distinct cultures were obtained. Unfortunately the growth of these cultures was not watched very closely, and I cannot say definitely whether the culture was originally white all over or whether there was a mixture of pink and white from the first. The white culture corresponds somewhat to the trichophyton

accuminatum in its appearances, but is smoother on its surface. Until I have watched its growth by further subculture I am not prepared to say whether it is the trichophyton accuminatum or a pleomorphic form of the pink culture.

So far as I can find from the literature, Sabouraud and Favera are the only two observers who have previously investigated the kinds of cultures obtained in ringworm of the nails. Sabouraud found trichophyton accuminatum and trichophyton violaceum; Favera obtained trichophyton violaceum and trichophyton rosaceum. To these must now be added the trichophyton crateriforme flavum found in my cases.

Treatment.—The treatment in these cases was difficult. Many of them were only seen once, so that I do not know whether the treatment recommended was ever carried out or not. All methods of treatment have for their object the softening and mechanical removal of as much of the diseased nail as possible and the application of some continuous antiseptic dressing.

The easiest method is to keep the nail covered with an india-rubber finger-stall after applying ung. hydrarg. ammoniat. night and morning. Of course the softened nail must be scraped away every day. One of the cases was cured by this method in 3 months, but in the majority of cases that method is quite ineffectual. A better method is Harrison's, *i.e.* in addition to the regular daily scraping a solution of pot. iodid., ℥j. in liq. potassæ, and aq. destill., āā ℥ss., is applied for half an hour on a piece of cotton-wool. It is then removed, and for the rest of the 24 hours a solution of hydrarg. perchlor., grs. iv. in spir. vini, and aq. destill., āā ℥ss., is applied on lint and the finger covered with an india-rubber finger-stall. These applications are repeated daily till the nail becomes black and so soft that it can be removed. In one case a cure was effected by the above method in 2 months. The reason for the failure in the other cases in which it was tried was that the patients stopped the applications as soon as the nails became the least painful. To get a good result the patient must be seen frequently, and the nails scraped by the doctor himself.

A new method which has given encouraging results is one devised by Dr Norman Walker. He applies Fehling's test solution on lint to the nail, covers it with a finger-stall, and leaves it on for a day or two. At the end of that time the whole nail is so soft and pulpy that it can easily be removed by forceps. After the nail is removed a solution of copper sulphate, grs. 10 in water ℥j., is

used as a continuous dressing. With this method good results were obtained in some of the cases; but whatever method is used long and painstaking treatment is essential for a successful result. When the disease is cured the nail grows in again without any deformity.

Favus Cases.—I have only seen 2 cases of favus of the nails. These were both in children with favus of the scalp. The appearance of the nails is very similar to that of tinea, but the nails are yellower in colour. A mass of fungus also tends to accumulate like a scutulum underneath the nail and raise it up. The diagnosis is made by finding the fungus microscopically, and the treatment the same as in ringworm.

The other fungus infection which I wish to mention occurred in a girl aged 22, in whom 3 nails had been diseased for 10 years. The nails were greenish-yellow in colour and splintering off. There was also some heaping up of the nail at places. Scrapings from the nails showed that fungus was present, but it differed altogether from the fungus seen in the ringworm and favus cases. The mycelium was very fine, and in long slightly branched threads. The spores were arranged in bunches, and were round or pear-shaped. Cultures gave a fluffy greyish-white growth. This culture is certainly not a ringworm or favus, but without further observation I am not prepared to say to what class of fungus this one belongs. The nails were removed in this case after being softened by caustic potash, but some weeks later the new growing nail was still found to be infected with the fungus.

Towards the expenses of this research a grant was made by the British Medical Association.

Discussion

Dr W. Allan Jamieson said he was sure they had all listened with great pleasure to Dr Cranston Low's interesting paper; it showed not only great observation, but very great pains in making all these beautiful casts and preparing the cultures. Up till quite recently the existence of ringworm of the nails had been almost denied; in former times it was said to be a very rare thing, and only recently its greater prevalence had been recognised. In the past they were far too ready to diagnose cases of fungus of the nails as eczema or psoriasis, and were not sufficiently careful to examine them with the microscope and liquor potassæ. It was not always easy to detect the fungus unless you took repeated parts

of the nail, the fungus was not universally present and was apt to escape detection unless repeated observations were made.

One interesting point was that it was always the large-spored fungus which was found, and that, as Dr Cranston Low had said, accounted for the rarity; the large-spored fungus in ringworm of the head was by no means common. It was interesting to see that the cultures all brought out the same fungus.

Treatment by Harrison's method was very tedious and caused a great deal of pain; he was glad to hear that Dr Norman Walker had found a modification of this treatment by means of Fehling's solution, and that in many cases it produced satisfactory results. Possibly further investigation would reveal a still more simple method of treatment. By using peroxide of hydrogen after softening the nail an altogether painless and satisfactory cure might be obtained.

Dr Norman Walker thanked Dr Low for his admirable paper and careful, painstaking research of what seemed to be an uninteresting condition. He confessed to having regarded fungus of the nails as rather troublesome, inasmuch as it was not a subject that very much was known about, and when faced with it in the middle of a clinique one was expected to say something about it; one used to put it down as a trophic disturbance, and this diagnosis was still made at times. Dr Low developed some years ago an active interest in this disease. He did not start by investigating ringworm of the nails; while investigating other conditions he came upon it. It was a remarkable fact, and he entirely agreed with what Dr Allan Jamieson had said, that they must have overlooked these cases before; he did not know what his diagnosis had been nor what Dr Allan Jamieson's had been, but in the records of the cases it was stated that the patient had been up before and that the disease had lasted for ten years and so on. Again, these cases were not confined to the lower classes, two of his patients were ladies who were art students. It was quite possible one might be confronted with such a question as: What was to be done with a schoolmistress who had ringworm of the nails? Over and over again recently he had seen cases which he at first thought were not ringworm cases but had turned out to be so, and had been astonished to find how frequent the disease was.

The treatment to which Dr Low referred rather surprised him; its discovery was an accident. He had used Dr Harrison's treat-

ment for a time, and had tried it on the scalp with very disastrous results—patients refused to continue it ; on the nails, too, it caused a great deal of pain and patients would not go on with it. He proposed to use sulphur of hydrogen, and intended to put some liquor potassæ with it, but knowing that it took at least twenty-four hours for medicine to come from the Dispensary, he thought of using Fehling's solution. It was remarkable how Fehling softens the nail, one could lift the nail off, and it caused less pain ; he recommended surgeons to use it.

He thought Dr Low was to be congratulated on a really sound, good piece of work.

The *President* asked if Dr Low knew of any cases in which ringworm had been communicated to others by persons suffering from ringworm of the nails ?

Dr Cranston Low said, so far as he knew, there had been no cases in which infection had been carried in that way. There were only two of his cases in which there was a direct history of infection—in one, the mother had been attending to her children and had got the infection, but it was just possible she might have had the condition previously and had infected the children ; in the other case the patient's brother suffered from ringworm of the beard and it was supposed that the infection had been communicated to her.

Dr Norman Walker thought that it was more likely that the patient with the nail affection had infected the brother, because ringworm of the beard brought the patient to a doctor very quickly.

Mr Gardiner mentioned a case in which the father had had ringworm of the nails for ten years, and two of his children had ringworm. It was difficult to see what the father would be doing scratching his children's heads, but he believed that the father had communicated the disease to his children. In all the cases the large-spored fungus was found and all were microscopically identical. The father had been treated by an English dermatologist. There was great difficulty in finding the fungus in this instance ; it was only after leaving the nail for half an hour in liquor potassæ that it could be detected.

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Meeting II.—November 30, 1910

Dr Byrom Bramwell, *President, in the Chair*

I. Election of Members

Lewis H. F. Thatcher, M.D., M.R.C.P. Ed.; Alexander Ernest Chisholm, M.B., F.R.C.S. Ed.; Norman Scott Carmichael, M.B., M.R.C.P. Ed., were elected ordinary Members of the Society.

II. Original Communication

ARTERIAL PRESSURE

By G. A. Gibson, M.D., LL.D.

My first duty is to express my great pleasure in having this opportunity of bringing before the Society some of the important facts relating to arterial pressure as an introduction to what, it is hoped, will be an interesting discussion. My original intention, when speaking to the Senior Secretary on the subject, was simply to bring a paper before you dealing with certain aspects of the subject which have been, during the last few years more especially, occupying my attention; but it will certainly be more interesting to us all to have an open discussion of the subject at large.

Definition of Terms.—Before entering upon the consideration of the different divisions into which the subject naturally falls, it is obvious that we must be at one in regard to the terminology to be employed. The *maximal*, or *systolic, pressure* is estimated by the amount of force which will entirely arrest the flow of blood in the vessel undergoing investigation. In regard to this point there is probably no difference of opinion whatsoever, except with reference to the relative importance of the resistance offered by the wall of the vessel—a matter which will engage our attention shortly. The *minimal*, or *diastolic, pressure* is estimated by the point at which the greatest lateral oscillation of the arterial walls is found. This is ascertained by the level at which the greatest systolic and diastolic movements of the column of mercury are

observed. This part of the subject has given rise to more difficulty than any other in the study of arterial pressure, and has led to wider discrepancies of opinion. Marey, for example, regarded the greatest oscillations as being an expression of the mean pressure. In this belief he was followed by Hill and Barnard, who expressed a similar opinion, but since the investigations of Howell and Brush we know that the greatest oscillations are found to indicate the diastolic pressure, their careful investigation having absolutely proved this point. The *mean pressure* is taken to be a point halfway between the maximal and minimal, while the *pulse pressure* is a term employed to denote the amount of difference between the maximal and minimal pressures. The use of this latter term —*pulse pressure*—cannot be commended, as it savours of ambiguity. The term, however, has become implanted in modern literature, and when that is the case it is difficult to modify the terminology.

The arterial pressure, as measured by our clinical methods, is the lateral pressure, or the pressure which is exerted on the walls of the vessels at a right angle, approximately, to the line in which the onward current of blood flows. The terminal pressure may be regarded as this lateral pressure, in addition to the velocity of the blood. The general conception is that the lateral pressure in an artery is about equal to the terminal pressure in the branch of the vessel immediately beyond the point at which the lateral pressure is estimated.

In the discussion of various problems connected with arterial pressure it is useful to take into consideration the co-efficient of pressure, as it provides us with a convenient method of estimating the validity of the circulation. The co-efficient of pressure consists in the pulse pressure, divided by the systolic pressure. Dealing with anyone in ordinary health, we may say (expressing the pressure in millimetres of mercury) the systolic pressure is 120, the diastolic pressure is 90, and the pulse pressure is 30. This gives us :—

$$\frac{\text{P.P.}}{\text{S.P.}} = \frac{30}{120} = \frac{1}{4}$$

which is the co-efficient of pressure. We may further assume that the pulse pressure, multiplied by the pulse-rate, is equivalent to the velocity ; while the systolic pressure, multiplied by the pulse-rate, expresses the work done. From this it is easy to calculate the efficiency of the heart. For anyone in perfect health,

in formulæ similar to those of Tigerstedt and Hirschfelder, we may state the condition of matters in the following way :—

$$\frac{\text{P.P.} \times \text{P.R.}}{\text{S.P.} \times \text{P.R.}} = \frac{\text{Velocity}}{\text{Work}} = \frac{30 \times 70}{120 \times 70} = \frac{2100}{8400} = \frac{1}{4}$$

gives efficiency of heart in health.

Let me, as a contrast to this, state what occurs in an instance of heart block :—

S.P. = 270 ; D.P. = 90 ; P.P. = 180 ; P.R. = 30 $\frac{180 \times 30}{270 \times 30} = \frac{5400}{8100} = \frac{2}{3}$

In a case of aortic disease the departure from normal is even greater :—

S P. = 160 ; D.P. = 80 ; P.P. = 80 ; P.R. = 90 $\frac{80 \times 90}{160 \times 90} = \frac{7200}{14400} = \frac{1}{2}$

Influences at Work.—The factors which maintain the arterial pressure must be briefly referred to. The first and greatest influence is the *energy of the heart.* Although many other causes are in operation in the maintenance of the arterial pressure, this is the most important. The heart still remains, as Harvey said, " the sun or centre of the circulation." If, for any reason, there should be an increased output of energy in the heart, the arterial pressure is augmented ; and if, on the other hand, there should be diminished cardiac energy, the arterial pressure will necessarily fall, other conditions remaining constant.

The condition of the *arterial walls* plays a prominent part in the maintenance of the pressure. When the arterial walls are in conditions of health, so as to permit of the uninterrupted passage of the wave of increased pressure produced by cardiac activity, and when the walls retain their power of elasticity (meaning the property by means of which, after any change of form, they return to their previous condition) the state of matters is most favourable for the circulation. With every pulsation of the heart there is a corresponding increase of pressure within the arteries, and some of this pressure distends the arteries, giving rise to potential energy which, during cardiac diastole, becomes kinetic. There has been a considerable amount of misconception as regards the condition of the arterial walls. In the first place, the term elasticity has been used with very different conceptions, but, taking it in the sense employed by Kelvin and Tait, we may hold that it is the property by means of which a body that has been distorted by any force returns to its original bulk and shape on the removal of

that force. In the second place, the resistance of the arteries to external pressure has been the subject of considerable debate within recent years. According to many authorities, as may be found expressed by Janeway, the resistance of the arterial walls to external pressure may be regarded as a negligible quantity; while, according to others (and here Russell may be regarded as the protagonist) the external resistance of the arteries is the most important factor in arterial pressure, as gauged by clinical methods. Here, as in many other matters, the truth lies somewhere between the two extremes, but it is certainly nearer the statement of Janeway than of Russell. The question can easily be solved by testing the resistance of the arteries by the methods employed by Tait in the investigation of distensible tubes. A piece of the brachial artery (which is specially selected seeing that it is the artery in which we estimate arterial pressure most commonly) is tied at its two ends to two pieces of glass tubing, and fixed, by means of these, inside a glass tube, with rubber stoppers at each end, through which the two pieces of glass tubing are inserted. By means of two other glass tubes a stream of water is caused to flow from a reservoir, while another stream of water is led through the artery. Each system, internal and external, is in communication with a mercurial manometer, and when water is allowed to flow through the two systems, the amount of resistance required to compress the artery can be accurately determined. It my own observations the amount of arterial resistance ascertained in this way has never exceeded 15 mm. Hg, but in the series of 49 cases carefully studied by Herringham and Womack they found two instances in which the pressure was above 30; one of these gave 33, and the other 34 mm. Hg. With these two exceptions, in no case was the obliterating pressure above 24 mm. Hg. It may be held that experiments upon arteries after death do not help us as regards sphygmomanometric investigations during life, but it is to be remembered that the tonus of the vessels does not cease at the moment of death; on the contrary, it remains for a variable period.

The *resistance in the arterioles* is, next to the cardiac energy, the most important factor in the production of arterial pressure; in fact, the resistance and the pressure are reciprocal. The resistance in the arteries is produced by tonus, which is the result of the antagonistic action of vasoconstrictor and vasodilator nerves. There is a great difference between the action of these two sets of

nerves ; the former are constantly in action, the latter are only brought into action when they are required. These agencies are always in a condition of variation, so that the general and local vasomotor tone may be regarded as perpetually undergoing alterations.

The *character of the blood* undoubtedly plays a prominent part in the arterial pressure. The *density* appears to me to be in a direct ratio to the pressure. The few observations which have been made by myself personally have been with the view of illustrating the condition of matters in splenomegalic polycythæmia. These observations have led me to the belief that the specific gravity and the pressure bear a direct ratio to each other. This appears to be the general conclusion of most observers. The *viscosity* of the blood might be thought to stand in the same relation as the density, and here again the few individual observations made by myself would lead me to that belief. A much larger series of investigations, carried out by Welsh at my suggestion, and shortly to be published, throws considerable doubt upon this matter, and it is one which will require careful investigation in the future. The presence of *chemical impurities* in the blood increases, for the most part, the pressure, but certain impurities bring about the opposite result. Whether these effects are the result of direct toxic blood on the arterioles or on their nerves is at present uncertain. The *quantity* of the blood in the circulatory organs must have some influence, and it might naturally be expected that increased volume would result in higher pressure. The vasomotor nervous system, however, has such ample powers of compensation that the quantity of the blood in itself has not, probably, such influence.

The local condition of arterial pressure in any part of the body is not merely modified by vasomotor activity, but also by mechanical interference. In other words, alterations in the *access of blood* bring about modifications in local pressure. In has long been known that any obstruction increases the proximal and lessens the diastolic pressure. The external pressure of an aneurism or a neoplasm, or the blocking of the lumen by embolism or thrombosis, reduces or terminates the pressure, and this often without any alteration in the condition of the wall of the vessel itself. A very interesting instance of this was under my care in the Royal Infirmary just about a couple of years ago. A man of middle age was admitted with aortic disease. The left arm furnished a beautiful instance of Corrigan's pulsation, while there was nothing of the kind to be

found in the right. When the pressure was tested in the two arms it was found that in the right arm, the systolic pressure was 74 and the diastolic 55 mm. Hg ; while in the left arm the systolic pressure was 130 and the diastolic 90 mm. Hg. The patient died somewhat suddenly, as the result of his cardiac disease, and after death a thrombolic obstruction was found at the origin of the right subclavian artery. The brachial arteries were carefully tested after death by Dr Rainy and myself, and we discovered that the resistance in each artery was practically the same, the figures being 11 mm. Hg in the right brachial and 10 mm. Hg in the left.

Methods of Investigation.—Amongst the methods of estimating the arterial pressure from the *physiological point of view* the earliest observations were made by Hales, who ascertained the height to which the column of blood could rise in a glass tube, connected with one of the larger arteries in the horse. At a later date the use of the mercury manometer was introduced by Poisseuille, and the method was rendered more available by Ludwig. Still more recently the introduction of the elastic manometer, such as the membrane of Chauveau and Marey and the metal spring of Fick, may be particularly mentioned. It is not necessary on the present occasion to dwell on these physiological instruments.

Attempts at the *clinical estimation* of arterial pressure have a respectable antiquity, and it is possible from the writings of Aristotle, Herophilus, and Galen to bring forward much of importance as regards this aspect of the study of the pulse. In the works of Galen the investigation of the pulse is carried out with what, in this age, appears to be excessive refinement. There cannot be a doubt of the extreme importance of educating the sense of touch in the estimation of arterial pressure, and in my own clinical teaching, after impressing upon those who give me the privilege of teaching them that their first duty lies in trying to ascertain, with as much accuracy as possible, the state of the arterial walls, the next place is given to the resistance within the vessels. Although, to my way of thinking, it is quite as scientific to attempt the estimation of the temperature in a case of fever by means of the unaided thermic sense of the physician as to try to gauge the arterial pressure by means of tactile sensibility, yet it must be admitted that we are sometimes inclined to trust too much to our modern instruments. It is therefore necessary, before teaching the use of such instruments, to educate our clinical clerks most carefully in the methods of estimating arterial pressure by means of the sense of touch.

The first attempts at the *clinical estimation* of arterial pressure by *modern instruments* were made by Vierordt, who, using one of his own sphygmographs, attempted, by attaching different weights to it, to estimate the amount of pressure necessary to overcome the arterial pressure. The first observer, however, who really brought the clinical estimation of arterial pressure within practical possibilities was von Basch, who was followed, at a somewhat later date, by Potain. Marey, Waldenburg, Hoorweg, Hürthle, Bloch, Mosso, and Oliver, during the last quarter of the previous century, applied themselves to the problems arising from the study of arterial pressure in man, and also paved the way for future advances; but it is not too much to say that von Basch and Potain deserve the credit of being pioneers in our clinical methods. The present period is entirely due to the introduction by Riva Rocci of the method of circular compression of a limb, so as to obliterate one of the larger arteries and estimate the effects by examination of itself or of one of its branches further from the heart. It may be said at once, without fear of contradiction, that, with the exception of those sphygmomanometers which apply this method, there is none which need be seriously considered. The others are of historic, but not of scientific, interest. Another point is that the only reliable instruments are those which are provided with a column of mercury. All the others, whether employing an aneroid, a column of air, or a tube of spirit, are not to be trusted, unless they have been standarised immediately before use by comparison with a mercurial manometer. Judged by these standards, the instruments of Marey, Hürthle, and Mosso, which embrace the extremities after the manner of the plethysmograph, and that of Gärtner, which estimates the pressure after the removal of the compression of a finger by the return of colour to the skin, as well as the instruments of Hill and Barnard, and of Oliver, cannot be recommended. On the other hand, the apparatus of Riva Rocci, Stanton, Cooke, Janeway, Martin, French, and Mummery are at once perfectly simple and absolutely reliable. In the last two or three years graphic records of the arterial pressure have been obtained. The sphygmomanometer of Erlanger, while based upon the same principles as that of Riva Rocci, was the earliest attempt to furnish graphic records of arterial pressure in clinical investigation. A tube connected with the column of air, leading from the cuff to the mercury of the manometer, communicates with a hollow rubber ball, which responds, by distending and contracting, to every

oscillation of the air and the mercury. This ball is surrounded by a glass bulb, leading by a tube to a tambour connected with a lever, which records the oscillations upon a smoked cylinder. These tracings furnish a complete record of the oscillations of the column of air, but do not furnish any record of the height of the column of mercury by which the pressure may be estimated. It is, accordingly, necessary to watch the level at which the mercury stands while noting the oscillations. The return of the arterial pulse at the wrist coincides with the commencement of the large oscillations, and the point at which the large oscillations suddenly begin to diminish gives an indication of the diastolic pressure. The sphygmomanometer recently introduced by me also takes advantage of the principle of circular compression in order to estimate the maximum or systolic arterial pressure, while it at the same time gives the minimum of diastolic pressure by the oscillations of the mercury. It has a mercurial manometer, the lumen of which is exactly that of the ordinary physiological kymograph. The air contained in the armlet can be increased, and the pressure on the limb therefore elevated, by means of a large syringe, and the pressure may be raised quickly or slowly according to requirements. By means of a valve the pressure may also be lowered quickly or slowly. A float rests upon the mercury, surrounded, as is usual in the physiological laboratory, by alcohol, and an upright rod of aluminium leads to a horizontal arm which writes on the revolving cylinder. In order to have the absolute zero, a fixed arm traces the abscissa upon the cylinder,which is driven by a clockwork placed horizontally, as in the instrument of Erlanger. The pulsations of the artery below the point of compression are recorded by means of a transmission sphygmograph. This consists of a tambour brought into contact with the brachial or radial artery, as may be most convenient, by a pelotte resting upon the vessel. This tambour is brought into communication by rubber tubing with another tambour, the movements of which are recorded on the cylinder simultaneously with the movements of the kymograph. The best tracing is obtained when the tambour in contact with the artery is larger than that connected with the recording lever, by means of which the movements are amplified.

As usually employed, the pressure is raised with one steady, forcible expulsion of the air contained in the syringe until a high level is reached—150 or 160 mm. Hg being sufficient under ordinary circumstances. The valve already set at the slow escape

allows the pressure to fall gradually. If sufficient pressure has been employed, the tracing from the artery, after a few oscillations due to the inertia of the mercury, shows an entire absence of all movements.

In the interpretation of the tracings there is one point which may always be depended upon with a reasonable degree of certainty; the point at which the pulsation returns in the vessel below the seat of compression is approximately the systolic pressure. This has been admitted ever since the observations of Vierordt and von Basch. The middle of the oscillation at this point is therefore chosen as the index of systolic pressure. It is perfectly true that it is not the end pressure. The top of the first wave which appears gives the maximum systolic pressure indeed, but it is the lateral and not the end pressure which is recorded, and therefore the first pulsation by the method of circular compression, as was shown by Masing, does not give the absolute maximum. The determination of the minimum or diastolic pressure is not such an easy problem. Marey suggested that the point at which the largest wing of the instrument occurred was an index of the mean pressure, and this was adopted and amplified by Roy and Adami. It has been proved experimentally, however, by Howell and Brush that this does not indicate the mean, but really records the diastolic, pressure. Masing, who obtained some tracings from the artery below the seat of compression, believes that the greatest movement of the sphygmographic tracing marks the diastolic pressure. This must, however, be an error, as the greatest amplitude of oscillation of the sphygmographic curve is very commonly found after the pressure in the armlet has been allowed to fall nearly, if not quite, to zero, and the results obtained by this method of estimation are unmistakably erroneous. My own method of obtaining the diastolic pressure is to ascertain where the greatest amplitude of oscillation occurs in the kymographic curve, and to take the middle point of this as the expression of the diastolic pressure.

A very beautiful modification of my sphygmomanometer has been introduced by Singer, who has reduced the size of the apparatus, and has applied to it an ink pen, so that records can be obtained without the troublesome necessity of smoking the paper on the cylinder.

During this same period, when our attention has been applied to the graphic investigation of arterial pressure, auscultatory determination has also been studied. Korotkow, Ettinger,

Bozowski, Krylow, Ehret, Fischer, and Gittings have, during the last five years, paid much attention to this subject, which is, both pathologically and clinically, of very great interest. These determinations are carried out with an ordinary sphygmomanometer of the Riva Rocci type, applied, in the usual manner, to the arm, and the brachial artery is carefully auscultated at a point about half an inch below the armlet. The arm is compressed in the ordinary way, and when the column of mercury is allowed to fall, on releasing the valve, a distinct sound, with the character of a thud, is heard. This denotes the maximum, or systolic, pressure. Variations in this sound follow, and at last, with still further lowering of the column of mercury, the sound altogether disappears. This is generally understood to correspond to the minimum, or diastolic, pressure. This method has proved of much interest in my own wards, as we have found in it a very definite way of estimating the acuteness of sensibility of the observer. When the same observer listens to the sounds, and, at the same time, applies his fingers to the arterial pulse, the audible and palpable phenomena occur at exactly the same moment of time. If, on the other hand, one observer listens to the brachial artery whilst another feels the arterial pulse, a discrepancy is perfectly certain to take place. It seems to me, from my own observations in a large number of cases, in hospital as well as private practice, that the auscultatory determination cannot replace the previous tactile determination. It nevertheless furnishes a useful additional method to our modes of investigation.

Possible Fallacies.—It may very pertinently be asked, Are there no fallacies in our modern methods? It seems to me that our new methods of investigation provide few, if any, sources of error. With a good and simple instrument the results which we obtain contain few possible sources of error in respect of determination, leaving at present out of account the question of interpretation. Riva Rocci and Henson have proved the accuracy of these methods on the dead body.

With all these instruments the influence of the personal equation is reduced to a minimum, and the question involves merely the variable acuteness of sensibility of the observer. This has been already referred to in my remarks on auscultatory determination. Russell, as has already been mentioned, believes that the wall of the artery under compression has a great influence on our results, and that degenerative conditions of the wall, in

addition to increased contraction of its muscular tunic, account for some our our highest readings. It must be remembered, however, that the middle coat of a large, is relatively much thinner than that of a small, artery, and my own opinion is that Russell mistakes the increased pressure produced by contraction of the arterioles for changes in the walls of the large arteries which we are in the habit of investigating. Henson, from a long series of observations, showed that the resistance of the vessel walls under ordinary circumstances was, on an average, equal to 3 mm. Hg, and that, even when they were much sclerosed, it was never higher than 20 mm. Hg. The observations of Herringham and Womack showed that in only 2 out of 49 cases the resistance was above 24. We may, therefore, until the contrary has been proved, assume that any changes in the vessels will not account, at most, for more than 20 or 30 mm. Hg.

It is clear that the limitations by which we are surrounded in attempting to estimate the absolute value of arterial pressure consist, to a great extent, in the difficulties by which we are beset in analysing the different factors maintaining the pressure. The relative amounts of cardiac energy and of vascular resistance are still, as was remarked by me in the Toronto discussion, a sealed book. A relatively low general pressure may be one which is nevertheless too high for the energy of the heart, while on the other hand a pressure which appears to be abnormally high may be one which is quite low in relation to the strength of the heart. The fact that spasm of the arterioles and smaller arteries has an important influence in modifying the pressure must always be borne in mind.

It must be added that when there is any very high degree of adiposity or any extreme amount of œdema, readings with the sphygmomanometer are rendered less easy and more doubtful. It is further to be noted that if there should be any liability to tremor, accurate observation is rendered less easy.

The pressure should always be estimated while the patient is in a position as nearly as possible horizontal, and, at the same time, resting comfortably. The limb which is employed for investigation ought to be absolutely at rest.

Arterial Pressure in Health.—The *normal limits* of arterial pressure require a brief notice. From numerous observations with our modern instruments by a large number of reliable observers, it may be assumed with certainty that the normal systolic pressure

in the young male adult does not exceed the limits of from 90 to 100 mm. Hg, while the diastolic is from 70 to 100. It is less in healthy women than in men of the same age, and still less in children. In healthy individuals variations in the arterial pressure take place without the presence of abnormal conditions, and must be accounted as normal. Daily fluctuations, probably dependent upon the condition of the whole system, are always found. The pressure usually reaches its highest level in the forenoon, after which it falls, frequently, however, manifesting another but smaller rise in the afternoon. Recognising that such changes exist, we will be wise if we always take our readings in any individual at the same hour.

When the pressure is studied by means of continuous tracings, smaller variations, both in the level of the pressure and the amplitude of the oscillations, may be observed. They are clearly analogous to the Traube-Hering curves seen in physiological tracings taken with the kymograph, and they undoubtedly depend upon changes in the influence of the vasomotor nerves.

The *external temperature* must exert some influence, but we still remain in want of sufficient observations upon the subject. So far the general result of the investigations which have been made (by Müller, for instance) shows us that when an individual is exposed to any considerable alteration of the external temperature the arterial pressure is apt to rise.

The *atmospheric pressure* exerts considerable influence over arterial pressure. Most of our knowledge upon this subject is due to Crile, who has shown that the external atmospheric pressure and the internal arterial pressure stand to each other in a direct ratio.

Posture exerts a certain amount of influence. There is not much difference in arterial pressure between the recumbent and the sitting postures, but there is a much greater difference between sitting and standing. It is the diastolic pressure which undergoes the greatest changes, so that in this way the pulse pressure is diminished in the upright position.

The *influence of food* is not constant. After a meal the pressure sometimes rises and sometimes falls. Diminution of pressure is easily accounted for by the diversion of blood into the abdominal viscera, but this may be balanced by reflex stimulation of the vasomotor system. After the contents of the alimentary tract have begun to enter the circulation there is certainly a tendency to elevation of the blood-pressure.

The question of food at once brings up the analogous subject of the effect of certain articles much employed in daily life. It is almost certain that the usual influence of *alcohol* (after a slight initial rise produced by increased frequency of the heart) is to cause diminished pressure. *Tea, coffee, and cocoa* have not yet received the attention which they deserve, and a series of observations upon these substances would be most helpful. In regard to *tobacco,* we well know that its effects upon the vaso-constrictor apparatus bring about a very great increase of arterial pressure, and this view entirely accords with a great series of observations made in France, showing that arterial sclerosis is frequently brought about by the excessive vascular contraction, resulting from the abuse of tobacco.

Muscular exercise produces remarkable effects. Gentle exercise has little, if any, influence in raising arterial pressure, even when it is continued for a considerable length of time. Violent muscular exercise is followed first by a considerable rise of pressure, and this is succeeded by a striking fall at a later stage. The observations of Gordon, carried out when he was my house physician, are sufficient to prove these facts.

Mental exertion has similar effects, but these vary with the intensity of psychical processes. This is a matter which has been tested very frequently in my clinique, both on myself, my staff, and my clerks, with results which are extremely uniform.

The arterial pressure certainly falls during *sleep* ; it is lowest in the early hours of normal sleep, rising gradually from that period until the hour of waking. Lewis Bruce, as well as Brush and Fayerweather, have brought before us most interesting observations upon this subject.

The *age of the individual* has considerable influence. Until middle life there is no great departure from the normal limits of arterial pressure, but after that period there is undoubtedly a much greater tendency to a higher level. Clifford Allbutt has devoted his attention to this subject, and to myself personally his teaching has been of the greatest benefit. Russell has criticised his opinions very freely, but to my way of thinking he has failed to shake the foundations upon which Clifford Allbutt's views are based. The real facts can only be ascertained from a very large series of observations. There are undoubtedly many individuals who retain comparatively low pressure during long lives, while in others a high pressure begins almost in youth. As a general

rule, however, increase of age brings with it elevation of pressure.

Arterial Pressure in Disease.—In attempting to give a summary of the probable modifications of arterial pressure in pathological conditions, it seems to me that it will be advisable, in the first place, to deal with alterations which mainly affect the circulation. It is impossible to do this, however, without bringing into the field of discussion a large number of external agencies connected with every part of the body, seeing that the circulation is not merely subject to purely mechanical alterations, but also to chemical changes, and perhaps even more subtle influences arising out of metabolic processes, while, at the same time, the heart and the blood-vessels show unceasing response to nervous influences, and the circulatory variations in turn react upon the entire organism. We must assume that the cardiac energy and the arterial pressure are reciprocal, while arteriole contraction and arterial pressure stand likewise in a direct ratio to each other. These general principles are necessarily subject to various modifications from changes occurring in any of the factors concerned in the physiological or pathological processes present in any given circumstances. There is a general correspondence, universally recognised, between the level of pressure and the cardiac conditions. When the arterial pressure is high the cardiac impulse is forcible, the second sound is loud, and even the first sound produced by the left ventricle may gain in its intensity. When a case manifesting such conditions is watched for any length of time, some increase in the size of the heart is made out by the ordinary clinical methods of palpation and percussion, as well as by the screen and the skiagram. In such cases, also, tracings obtained with the capillary electrometer, or with the string galvanometer show us, as has often been observed by myself, that the electromotive changes are excessive ; but, in addition to such results, there are some even more characteristic effects arising from the relations of the arterial pressure and the cardiac energy. No one could have been more sceptical than myself in regard to the possibility of aortic diastolic murmurs without valvular disease, it having always appeared to me that the origin of the aorta was so resistant as to preclude the possibility of stretching from stress leading to a strain. Some years ago, however, the stern logic of fact caused me to recognise that my views had been based upon insufficient experience, and in a paper in the *Edinburgh Medical Journal* some cases of diastolic aortic murmur

without valvular lesions were published. In these cases the diastolic murmur appeared, disappeared, and reappeared, according to the condition of arterial pressure. These cases, however, were surpassed in interest by a recent experience in my ward. A man of middle age was sent in on account of aortic disease, and on admission it was found that a very musical diastolic murmur could be heard by anyone standing at the foot of the bed, *i.e.*, about four feet from the heart. This murmur, as usual, was louder on standing than on sitting, and yet more so than on lying down, while after walking round the ward it became still further exaggerated. His arterial pressure was found to be 95 diastolic and 145 systolic on admission. After a few days the murmur became so much diminished in intensity as to lose all of its musical character, and it could only be heard on auscultation with the stethoscope. After a few days more rest the murmur absolutely disappeared. It occurred to me that it might be of interest to find out the relation between the arterial pressure and the murmur. After quiet rest in bed the maximum arterial pressure was found to be 115, and with this pressure no murmur was audible ; with gentle exertion the arterial pressure rose to 130, a soft, aortic, diastolic murmur developed. After some strenuous exertion, such as walking quickly three times round the ward, the maximum arterial pressure rose to 145, and the murmur became so loud and musical that the patient himself could hear it. It was obvious that in this case there was not merely stretching of the fibrous and elastic tissues forming the aorta, but also of the muscular fibres surrounding the aortic orifice.

Entirely analogous results have been obtained on investigating the pulmonary artery, and the pulmonary diastolic murmur of high pressure described by Steell, Duckworth, Barr, and myself is undoubtedly produced in the same way. Keith has shown us that a loss of tone in the muscle fibres surrounding the mouth of the vessel allows stretching of the fibrous tissue.

Lower arterial pressure is, in general, associated with feeble cardiac impulses and short, high, weak sounds. The conditions in which this group of phenomena is found are frequently observed in surgical practice, and the work of Cushing and Crile demand our most careful attention.

Let me now direct your attention as briefly as possible to some conditions of arterial pressure in general and special affections. Amongst the *general diseases* let us take, as an example of the *infective* type, typhoid fever. Upon this subject the results of all

observers are wonderfully uniform. In the overwhelming proportion of cases of typhoid fever the arterial pressure is reduced. The pressure begins to fall during the first week, and continues to fall during the second, third, and fourth week. During the fifth week it begins to rise, and gradually regains the normal. The most important statistics upon this subject are contained in the observations of Crile. From the modern arrangements in our Royal Infirmary few opportunities have been afforded me, during the last few years, of watching cases of typhoid fever, but the few observations made in my wards entirely accord with Crile's results.

Amongst *toxic* conditions let me cite the affections due to lead. In all the cases which have been under my observation during the last few years the pressure has been elevated, and the various symptoms stand in a direct ratio to the rise of arterial pressure. This is the case in regard to the digestive, muscular, and nervous symptoms.

Lastly, amongst such general diseases there can be little doubt that *metabolic* disturbances, in which the arterioles are subject to the baneful effects of imperfect tissue changes, are followed by high arterial pressure. A very large number of conditions, which we sum up under the heading of chronic gout, are associated with high pressure, and pave the way, by means of it, for the structural alterations in arterioles and arteries affecting the entire organism. Such conditions are of everyday experience.

Turning now to *special diseases*, let me, in the first place, refer to affections of the *vessels*. There cannot, in this age, be any doubt that, without any structural alteration in the circulatory organs, including heart and vessels, there are conditions in which periods of pressure changes, both low and high, occur through the influence of the vasomotor system. To such conditions Pal gives the name of *vascular crises*. They are attended by alterations in heart beat and pulse wave, and accompanied by modifications in the amount of every secretion, as well as in the appearance of the skin, and they give rise to very considerable disturbance of the nervous system, particularly in its subjective aspect. Such crises are frequently produced by causes of purely nervous character, but they are also produced by various poisons circulating in the blood. They are apt to lead, in time, to structural changes in the arterioles and arteries, particularly in the myocardium and in the kidney. Such conditions frequently manifest all the features of vicious

circles, closely studied lately by Hurry, as the alterations in pressure are very apt to lead to nervous instability in varied forms. While crises of high arterial pressure are more common than those manifesting low pressure, the latter condition is also sometimes found as a critical phenomenon, and here again the tendency to a vicious circle is apt to manifest itself, as low pressure frequently brings about nervous depression. The arterioles and small arteries, therefore, may be regarded as subject to many influences leading to alterations in pressure, which bring in their wake undoubted pathological conditions.

When we approach the subject of *arterial sclerosis* we find a general consensus of opinion that endogenous poisons, no less than those which are introduced from outside, produce definite arterial degeneration. One important point is concerned with the question whether high pressure leads to arterial sclerosis. There can be no doubt that the question must be answered in the affirmative, and amongst many workers upon this subject it is a duty to cite Clifford Allbutt, Savill, and Russell. The former of these observers has indeed classified out a series of causes under the categories of toxic, hyperpyetic, and involutional, but he frankly admits that there is no sharp dividing line between these classes. That this is far from being a solution of the whole problem must be obvious, when we consider the fact that there is frequently arterial sclerosis without high pressure. In 506 cases Groedel found 35 per cent. in which there was no elevation of pressure, while Rudolf, in collaboration with Ellis and Robertson, found that the pressure was only above normal in 50 per cent. of well-marked cases of arterial thickening. Such facts have been under my own observation for many years, and one of the most striking corroborations of this statement is to be found in the observations of Elliot Dickson upon the arteries and pressure in miners, where, with extremely common arterial changes, there is seldom any modification in arterial pressure.

The relation of *angina pectoris* to arterial pressure forms a somewhat complex subject. While there is a variety of angina pectoris of occasional occurrence which seems to be entirely due to spasm of the arterioles—the angina pectoris vasomotoria of Landois and Nothnagel—and while in a great many cases of organic angina pectoris the attacks are preceded and attended by rise of arterial pressure, due to spasm of the arterioles, there are numerous instances in which the arterial pressure is below normal. This is a fact to which attention has been more particularly called by Morison.

It is, nevertheless, in such instances particularly that the pressure, although below normal, may be relatively too high for a weak and failing heart, and the caution already expressed should, in this place, be once more recalled. It is a well-known fact, to which Mackenzie has more particularly invited our attention, that in cases of arterial sclerosis there is a greater tendency to spasm of the arterioles than in ordinary conditions. Every observer who looks into the matter with an open mind must be struck by the enormous variation in the appearance of the arteries when a spasm of the arterioles is present, as compared with their condition in the absence of a spasm. A few years ago Pal showed that vessels which were almost straight with a pressure of 95 became highly tortuous when the pressure rose to 200 mm. Hg.

Turning to the heart, we find in *acute pericarditis* an affection with a great tendency to bring about a lowering of arterial pressure, through its weakening effect upon the myocardium, and it is one of the affections in which a favourable or an unfavourable prognosis may be reached, according as there is little or considerable lowering of the pressure. All the recent cases of pericarditis which have crossed my path have made excellent recoveries, and on looking over their records, the striking feature is that in no case was the arterial pressure below 125 mm. Hg. When the pericardium is distended with fluid, as the result of an acute attack, the arterial pressure falls to a level which is alarming, and which is one of the surest indications of the necessity for active intervention. In the case of *acute endocarditis* there is likewise a liability to a fall of arterial pressure, and here, again, the prognostic significance of the arterial pressure canot be overestimated. Amongst the various *valvular lesions* there is an immense series of different pressure results. In most of them, until fatal cardiac failure steps in, there is but little interference with arterial pressure, so wonderful are the powers of compensation possessed by the circulatory system. In aortic disease there is a most striking instance of remarkable divergence between the systolic and the diastolic pressure. This has been already referred to in the introductory remarks upon the co-efficient of pressure and the efficiency of the heart. It is very common in aortic disease to find a systolic pressure of 170 and 180, while the diastolic pressure may only be from 70 to 80. Even greater variations of pulse pressure have been recorded. Another interesting point in aortic disease, with insufficiency, is that the difference between the arterial pressure in the arms and legs is much greater

than in health. To this point attention has been particularly directed by Hill and Hare. It has been suggested by the former that the condition is a reflex one, intended by Nature to prevent cerebral anæmia. Other valvular diseases produce comparatively little alteration upon the arterial pressure, except in so far as they bring about alterations in the efficiency of the myocardium. Diseases of the *myocardium* yield widely different readings; in fact, the results entirely depend upon whether the myocardial troubles are due to primary alterations in the heart muscle, or whether they are secondary to disease elsewhere. In such affections as myocarditis, following upon diphtheria or other acute disease, the arterial pressure, in general, falls. The same occurrence takes place in the fatty degeneration attendant upon any grave disorder of the blood. On the other hand, in fatty infiltration the arterial pressure is frequently high, simply because in such conditions impure blood in the arterioles causes increased resistance. Needless to say, when the myocardium is undergoing hypertrophy, to compensate for increased resistance, the pressure is high. One of the most interesting of all myocardial conditions is to be found in heart block, in which, apparently from the long intervals between the ventricular systoles, there is in most instances a very high systolic, and a very low diastolic, pressure. Attention was called to this fact by myself about five years ago.

In affections of the *respiratory system* there are great variations of arterial pressure. For the most part, in asthmatic conditions there is a tendency to high pressure, while in pulmonary tuberculosis, as a general rule, the pressure is low. In pneumonia there are very different levels of pressure in individual cases. In the remarks on prognosis some useful indications will be brought forward in connection with this subject.

The arterial pressure in *glandular diseases* constitutes one of the most interesting subjects in modern medicine. In myxœdema, as is universally recognised, the pressure tends to be considerably above the normal; in exophthalmic goitre, although in general the pressure is below the normal, this fact is not of universal application. As the vasomotor control of the arterioles appears to be in a more unstable condition than in most diseases, it can be no wonder that the pressure is very variable. Addisonism furnishes one of the most fascinating problems in its relation to arterial pressure In the fully-developed condition the arterial pressure is always low, because in it the medullary portion of the suprarenal body is de

stroyed, and the production of pressor substance is accordingly annulled. There may be a deep discoloration of the skin without alterations in arterial pressure, and in these cases we may assume that the cortical portion alone is diseased ; on the other hand there may be profound depression, without discoloration, but with great reduction of the arterial pressure, and in such cases it is certain that the medullary portion of the gland is that which is affected. To these conditions attention has, within the last two years, been particularly directed by Rolleston and myself. Acromegaly sometimes gives high and sometimes low pressure. Here again the different effects may be correlated with the particular portions of the pituitary body which are implicated.

Amongst affections of the *kidney*, in acute nephritis, the pressure is in general elevated, but this is not so universal as is commonly supposed. In many such cases the pressure is really below normal. It has often seemed to me that the state of the pressure may vary according as the process affects the glomeruli or the tubules. This, however, is a subject upon which observations are greatly wanted. In most instances of chronic nephritis the arterial pressure is high, and it is particularly high in most instances of uræmic poisoning. The highest pressure which we meet with is found in chronic interstitial nephritis, with arterial degeneration and cardiac hypertrophy, especially (as was shown by Hasenfeld and Hirsch) when the splanchnic arterioles are implicated. In waxy degeneration of the kidney the arterial pressure is always low, unless the process is accompanied by a high degree of interstitial nephritis.

The relations which exist between alterations of arterial pressure and disturbances of the nervous system are of the most interesting description, seeing that a large number of nervous affections are produced by changes in arterial pressure, while, on the other hand, nervous disorders frequently bring about very definite modifications in the arterial pressure. In a considerable number of cases of adolescent insanity, and more particularly in precocious dementia, the arterial pressure is extremely low. The recent work of Rae Gibson has thrown a good deal of light upon the connection between the two conditions, and the facts which he has brought forward have been fully borne out by a few cases under my own care. In cases of melancholia there is, as a general rule, a pressure rather above the normal, while in mania, on the other hand, the pressure tends to be subnormal. In most of the painful affections, such as neuralgia

and the crises of tabes dorsalis, the pressure is almost invariably above the normal during the attacks of pain. Pal believes that in such cases the cause of the pain is a vascular crisis. Possibly the most remarkable series of phenomena appearing on the borderland of circulatory and cerebral changes is to be found in the facts connected with cerebral hæmorrhage. It cannot for a moment be doubted that the essential factors in bringing about cerebral hæmorrhage are structural changes in the arteries and increased pressure within them. These points are, however, eclipsed in importance by the clinical observations that after a hæmorrhage has occurred the arterial pressure goes on steadily rising, and the result of the beautiful series of investigations by Harvey Cushing is to show that the object of this high arterial pressure is to maintain the circulation in the vital centres at the base of the brain.

Arterial Pressure in Prognosis.—From the point of view of prognosis the study of the arterial pressure is of the greatest practical importance. In *acute disease* the relation between the rate of the pulse and the height of the pressure is of the highest value. In an address given by me some years ago the rule was enunciated that in cases of *pneumonia*, when the pulse-rate per minute does not exceed the height of the pressure in mm. Hg, the outlook is hopeful, but that when the converse occurs the prognosis is grave. Every observation which has been made since the date of that address has more firmly convinced me of its value. It has recently been emphasised in a most interesting lecture by Hobart Amory Hare. Within recent years pneumonia is the acute disease which has been most frequently under my observation, but it seems to me quite clear that some such formula might well be applied to other acute affections. In almost all of them, as has been stated, there is a tendency to a fall of the pulse-rate. In all of the acute diseases, if there is too great a tendency to fall of pressure, there must, in the mind of the observer, be considerable anxiety. Acute *nephritis* furnishes an interesting example of a disease in which the condition of pressure is of use from the point of view of prognosis. It has already been remarked that in many cases of this affection there is no increase, and that a decrease may even be present. If there is a considerable increase the prognosis is less favourable than when there is no rise at all.

Amongst *chronic diseases* most instances of anæmia result in diminished pressure, but this is by no means to be regarded as a

general rule, as chlorosis has frequently increase of pressure. With the exception of such cases, it may be said that the lower the arterial pressure the worse the outlook for the patient. The same is true in *cachectic* conditions and in *Addisonism*.

In chronic *nephritis* the arterial pressure is, in general, above the normal, but the degree varies according to the variety of the affection. In chronic *parenchymatous* nephritis, resulting in the *large white fatty kidney*, the arterial pressure is usually elevated, but by no means to the same degree as in interstitial nephritis. An interesting point, bearing upon the prospects of the patient, is that a rise of pressure above what has been found to be the normal for the case is very commonly the herald of uræmia. In chronic parenchymatous nephritis of the type ending up in the *small white granular kidney* the pressure is considerably higher, but here, again, a sudden rise is apt to portend the onset of uræmia. It is in chronic *interstitial* nephritis—the type which terminates in the *small red granular kidney*—that the arterial pressure reaches such enormously high readings, and it is in such cases that the prognosis is so largely dependent upon the influence of remedial measures upon the pressure. In severe cases, with cardiac, respiratory, and nervous symptoms, as well as extremely high arterial pressure, we are sometimes absolutely unable to influence the course of the disease to any appreciable extent.

Treatment of Changes in Pressure.—The last subject to which attention must be directed deals with the treatment of the conditions in which the arterial pressure is abnormal. In order to preserve the arterial pressure as nearly as possible at its normal level every method by which the general health can be best maintained must be adopted. Attention to the skin, both by baths and clothing; regulation of the diet, both as regards fluids and solids; care of every eliminating channel; and sufficient muscular exertion, together with adequate rest—all such matters must be taken into the most careful consideration. It must be obvious that there is no absolute rule to be employed in cases of departure from the normal; each instance must be judged and treated in accordance with its particular nature. When the arterial pressure is too low, as in acute diseases, it may be necessary to use digitalis, or strophanthus, or strychnine, or suprarenal substance, or its active principle, adrenalin. When, on the other hand, there is a great tendency to high pressure, the nitrites, the iodides, the hot pack, the vapour bath, and even venesection may require to be adopted,

along with drugs which increase the action of every channel of elimination.

Such are the points connected with arterial pressure which seem to me to be those from which a most interesting discussion may arise, and upon which the Society will certainly wish the opinions of the members.

Discussion

Dr Wm. Russell said in the ten minutes allotted to him he would attempt as clearly as possible to indicate what he regarded as the central phenomena in the important question before the Society. He was sure Dr Gibson would understand that it was from no disrespect to him that he avoided much of what had been said. He would begin at the extreme end of the phenomena and mention a case which was at present in his ward in the Royal Infirmary. The patient came in with a pressure of 295 mm. of mercury. With rest in bed and treatment it fell to 165, showing a fall of 130 mm. of mercury; this pressure swung back again to 180 and kept fairly steadily between 180-210; but while still in bed, this patient developed a brachial pressure of 70 mm. greater than this, associated with symptoms which it was impossible to enter into here. This rise was invariably associated with a definitely palpable contraction of the brachial artery, diminution of its size and hardening of its wall. There was no question as to what took place in the brachial artery, because the common statement, which all of them had been long familiar with, was that when pressure rose the periphery is constricted, but unfortunately the periphery had been regarded as capillaries and arterioles.

Nine years ago, before this Society, he had described a hypertonic contraction of the arteries. At that time he had not investigated farther back in the circulation than the radial artery. When the modifications of the Riva Rocci instrument came into use, and he began to use them, he found it was necessary to go farther back from the radial to the brachial artery. He had found that the brachial artery was one which was often readily palpable, and the condition of its walls readily discovered. On post-mortem examination he found that the brachial artery had a strong muscular coat which frequently underwent great hypertrophy, and the wall therefore became thickened. He had in his possession brachial arteries like pieces of caoutchouc, and yet the contention was that these thickened brachial

arteries have nothing whatever to do with the compressibility of the artery. It was difficult to realise how it was possible to contend that this was so. After death an artery like that varies enormously ; it becomes like a piece of thick cloth ; it is no longer the elastic tube with its tonic and hypertonic muscular coat. It seemed to him that that was the explanation of the fact that such eminent clinicians as Sir Clifford Allbutt and Sir Lauder Brunton talk about blood-pressure and heart-power, absolutely ignoring this condition of the vessel wall.

To-day he had read Janeway's latest contribution to this subject ; it had just been published, and he went the length of maintaining that the calcified artery has no effect whatever on the compressibility of that artery. He would leave it to them to consider whether this was a reasonable position and one which clinicians could really accept.

Going on to explain another change that the thickened brachial artery undergoes, Dr Russell referred again to the patient with a blood-pressure of 200. When the brachial underwent contraction the pressure was necessarily thrown back upon the aorta. Now there had been a difficulty here, and it was caused by the fact that there are certain cases with thick arteries which do not give a high reading. He had recently had a man in his ward with calcareous radial arteries and sclerosed brachials, in whom the brachial artery was not only thickened but dilated ; it had a thick wall, but it also was a big vessel (shown on blackboard), and whenever one got a tube like that it was easier to constrict than a tube like that (demonstrated). What had happened in this man ? This man, a moulder by occupation, had a big hypertrophied heart and he died from heart failure ; his lungs had become œdematous ; he was very rapidly pouring out fluid into both pleuræ, and yet he died with a pressure of 140 mm. of mercury. That is not a high pressure, but that man's heart was not working—if you take 120 as the average power of the heart—a quarter of the average. The forearm gave a reading of 30 mm. higher than the upper arm owing to the calcareous condition of the wall. Yet we are asked to believe that the arterial wall is negligible.

With their permission he would now go to the other extreme, namely, the state of affairs in calcareous vessels where one is dealing with high pressure—to him a more interesting part of the subject is the conditions where there is a relatively low pressure. He would illustrate his remarks by a patient who was convalescing

slowly from an acute pericarditis with a distinctly feeble heart. In this patient, as long as the pressure kept to 120 or 125 mm., everything went on pleasantly and there was no discomfort, but if from constipation or unwise feeding or from mental disturbance the vessels become hypertonically contracted—it is a fact that they do become hypertonically contracted, for the contraction can be seen and felt taking place in the brachial artery—the pressure rose from 140-145 when lying in bed. Constriction of the periphery had occurred, which includes the brachial artery. When the brachial becomes constricted, it becomes thickened as the result of the hypertonic contraction, but the rise of the pressure takes place behind this in the arch of the aorta. He could not, and no one could, say what the aortic pressure was. As soon as this rise in aortic pressure occurred the feeble heart was embarrassed.

The essential clinical fact was the constriction of the arteries up as high as the brachial, and it is that constriction which is being read ; there are no means of estimating how much pressure that constriction has thrown back upon the aorta, and it might be quite easily over-estimated, because, as Dr Gibson pointed out, the heart very readily strikes a balance and recovers itself.

Another case of the same kind was that of an old gentleman, aged 80, suffering from angina minor. He had a blood-pressure of 140-145 whenever he had an attack of angina minor ; these attacks were associated with a palpable and definite tightening up of the radials and also the brachials ; and whenever the pressure could be reduced to 120 there was no further heart trouble, and the radial and brachial arteries became normal. It was necessary to determine what would keep the pressure down, and in that particular case very small quantities of iodide of potassium were required to steady it and prevent the contraction. The question of iodide Dr Edwin Matthew would no doubt be able to say more about, but one plain fact was that in this old gentleman's case the pressure was 120 in the soft relaxed (tonic) vessel, but when the brachial became hypertonic it went up to 140. The pressure was thrown back on the aorta and the feeble heart was embarrassed. A high brachial reading means a rise in aortic pressure. No one questioned that ; all he would like to say is that we were estimating not the pressure in the aorta, but the compressibility of the brachial. The difference in the readings was the result of hypertonicity. The symptoms were those of heart embarrassment and was due to a constriction of a much wider periphery than had hitherto been recognised ;

this constriction threw the pressure back on the aorta and therefore a greater strain was put on the left ventricle.

Dr Rainy directed attention to certain physical aspects of the subject which were apt to be overlooked in purely clinical discussions.

He pointed out that, in those methods of determining blood-pressure which depend on the use of an armlet, one had to assume that the tissues between the armlet and the vessels are so plastic that they may be practically considered to transmit the pressure in the same way as a sac of fluid would do. This assumption that the rigidity of the tissues is negligible is probably justified in ordinary cases, but, as Janeway and others have shown, there are certain conditions in which it is untrue. If, for instance, œdema is present, the method of armlet compression is quite unreliable. A not unreasonable suggestion for this is that the tissues, in consequence of the tension produced by the œdema, become more or less rigid, just as a pneumatic tyre gets rigid when fully inflated, and thus interfere with the transmission of the applied pressure.

The question of the allowance which may have to be made for resistance due to rigidity of the vessel wall appears at first sight to be a more difficult one, but, fortunately, blood-vessels remain alive for many hours after somatic death, and further, they can be maintained in life after they have been removed from the body. There is thus no difficulty in ascertaining the influence of this factor by experimental methods. Recent work by Stevenson has shown that transfusion of a vessel with a fluid which acts as a vasoconstrictor can induce a condition of hypertonicity, and by a simple piece of apparatus the difference between the external and internal pressures when the vessel is just flattened by the external pressure can be ascertained. The difference between these two obviously gives the exact amount of resistance to compression which such an artery offers. Stevenson's results show that there is a wide degree of variation within physiological limits. Roughly, in ordinary cases it will be found to be less than 10 per cent., and even where there is marked hypertonus it rarely exceeds 30 per cent., although in a few extreme instances it proved much greater. One must therefore acknowledge that the vessel wall does affect the readings, but not to an extent which can materially vitiate the observations in the immense majority of cases.

He further pointed out that the blood-pressure in all the larger arteries is very nearly equal to that in the aorta, and that when one remembers that the resistance to the flow of ordinary fluids through tubes varies inversely as the fourth power of the diameter, it becomes obvious that practically the whole fall of pressure takes place in the arterioles and capillaries.

He also directed attention to the fact (which appears to be too much disregarded) that it is merely an assumption, and in many cases an unwarrantable assumption, to say that disappearance of the pulse coincides with closure of the vessel, for, as a matter of fact, the pulse disappears before the vessel is obliterated, as can be shown alike by experiment and by the application of mathematical formulæ.

In conclusion he referred to some of the earlier mathematical literature on the subject, and especially to two most valuable articles by Dr Thomas Young, one of which was his graduation thesis, the other the Croonian Lectures which he delivered at a later period of his life, whilst in Germany the work of the brothers Weber, both on "Wave Motion" and on "The Application of the Study of Wave Motion to the Pulse," remains a standard authority to the present day.

Dr Cargill Knott said it was difficult for the mere physicist to speak, but some remarks Dr Rainy let fall require a little supplementing—he was not quite sure that he understood them. There is no doubt that the strain is very much greater in the large tubes. Then there was the question of the way in which the pulse falls off along these tubes. He always thought—he might be wrong—that the fading away of the pulse towards the periphery was due really to viscosity acting in the fine tubes. He had made an experiment, which Dr Russell had expounded to him some years ago, namely, when the arteries in the arm are compressed, the compression closes the pulse at the wrist. That seemed to show—though Dr Rainy might be right—that a resistance had been produced there which was comparable with the resistance in the smaller arteries where the pulse is not at all conspicuous. It was a difficult question how that would affect the whole circulation; the whole circulation is affected by all those various forms of tubes, and the fact that by this constriction you destroy the pulse brings the arteries of the arm into somewhat the same category as regards the smaller arteries. In that case it might be that both points of view are sound enough,

but he would be sorry to be dogmatic on the question. It is much too delicate a thing for a pure physicist to say much about; the physicist pretends to know nothing beyond pure physics. He might be speaking rashly, but that was what struck him when hearing Dr Rainy, and he thought it possible that both Dr Rainy and Dr Russell were right.

Dr Elliot Dickson said he was not going to give any mathematical calculation, but as a country physician who had done some work with regard to this subject he should like to say something about arteriosclerosis in apparently healthy men. He and his brother, Dr Arnott Dickson, had taken a series of 500 miners who were apparently healthy—they did not complain of any symptoms and were thus preferable to ordinary patients. They took the blood-pressure by the Riva Rocci instrument in these 500 men of all ages, and found that in no less than 456 the arteries were thickened. Out of 116, 100 had easily palpated arteries. Of the 500 only 2 had atheroma, and they were old men. Only 44 out of 500 had radial arteries which could not be palpated. The blood-pressure was 140 mm., or one in 6 per cent. of the cases, and in each of these the urine was tested for albumen, with a negative result. In 469, or 94 per cent., the blood-pressure ranged within normal limits; all had thickened arteries; in some of the cases this was very obvious, especially in men with thin arms, where the arteries could be tested all the way up to the axilla. Over and over again the brachial was traced right up to the axilla; it could be picked up between the fingers and thumb and rolled about; it felt like a lead pencil. Tracings had been taken of the brachial artery, by Dr Gibson's kindness, such as had been mentioned, showing rates of 120-118, and one of 95 mm. of systolic blood-pressure, so that whatever increased blood-pressure is due to, there can be no doubt that thickening of the brachial arteries is at least not the only factor. No doubt with thickened arteries there is a tendency to increased blood-pressure, but that was more evident when taken in connection with increase in age of the patient. In arteries of the same degree of thickening, so far as could be detected by the finger—and after considerable practice one could tell pretty well—there was as great a variation as 85-165 mm. when the pressure was taken on three different occasions in a youth,of 18. That whatever the cause it cannot be entirely due to the resistance of the arterial wall. The great point about this series of cases is

that the men were healthy and had nothing wrong with their circulation.

There were many cases of hypertonic contraction in men who came into the surgery to have teeth extracted; after that slight operation, as was to be expected, the pressure was 20 mm. higher; on testing the men on a second occasion the pressure was found to be 20 mm. less than before.

So far as the cause of the thickening of the arteries in this series of cases goes, it is rather difficult to explain. All the cases have thickened arteries with low or normal blood-pressure. They correspond with what Clifford Allbutt calls toxic arteriosclerosis. The toxin in this series of cases has to do with conditions of work underground. He had known and tested cases where the arteries were perfectly normal and where thickening developed within a year after working underground. The nature of the toxin is somewhat difficult to ascertain, but probably it has something to do with the ventilation underground. There is an enormously high percentage of carbonic acid gas in coal mines; .7 per cent. represents good ventilation from a coal-miner's point of view; .04 above ground. Whether that was a factor in producing thickening of the arteries or not it was impossible to say.

Dr Oliphant Nicholson spoke of blood-pressure in regard to pregnancy.

Dr Shennan said he rather hesitated to enter into the discussion, but one or two things mentioned had suggested several points to him. Firstly, as to the terminology. The terms atheroma, arteriosclerosis, calcification of the arteries, had been used; probably the best term was arteriosclerosis, because it included all the others. It might be taken that high blood-pressure was the prevalent end of this condition; the usual conditions found post-mortem being arteriosclerosis, thickening of the arterial wall associated with atheroma, meaning localised thickening of the wall. One point in connection with the calcification of arteries is that in thickened arteries, which are also calcareous, there is a constant feature to be noticed, namely, that the whole arterial circulation is not affected. In the portions of the arteries corresponding to the flexures of joints, the calcareous deposit is not seen. That can be verified by X-ray photographs; one can tell where the flexure is, such as the knee joint, the elbow joint, the groin, by the fact that the

vessel is still quite pliable, the parts between the joints being resistant.

With regard to the quantity of blood. He had recently been watching post-mortems in Münich; what had struck him as most common was aortic regurgitation with thickening of the arteries and cirrhosis of the kidney. There is a general idea that it is the quantity of fluid (beer) which is the cause; if in place of the quantity the toxic effects of the beer are specially to be taken into account, it was a terrible indictment on the beer.

With regard to the viscosity of the blood, he had noticed in one or two cases of polycythæmia that the heart had not been hypertrophied at all; in one case the coronary arteries had been extremely atheromatous, in fact a fine thread could scarcely be passed through the vessel. The hypertrophy was an interesting point, because Zétulle had stated that great hypertrophy may occur although the left coronary was practically impervious. That there was some arterial communication between the coronory arteries was generally allowed.

Dr Gibson referred to interstitial nephritis and chronic parenchymatous nephritis; were they to assume that in all forms of chronic nephritis, and many people included with these the subacute forms, high blood pressure is always present? Does chronic nephritis include the small red kidney and the large white kidney? He himself distinguished the two conditions; he recognised a distinct small red kidney due to chronic nephritis, and another red kidney due to primary arterial disease. In dealing with post-mortem examinations he was inclined to think that in renal disease the result of arteriosclerosis, hypertrophy of the heart is distinctly greater, on an average, than in other forms of chronic kidney disease.

With regard to amyloid disease and chronic nephritis. Amyloid disease, it was said, was always associated with low blood pressure, chronic nephritis with high blood pressure; but the curious point is that one hardly ever meets with pure amyloid disease of the kidney; it is always associated with chronic interstitial nephritis or chronic parenchymatous nephritis. It was rather a nice point to reconcile these statements.

Dr Edwin Matthew said that many drugs had been recommended in past years as successful in high blood-pressure associated with arteriosclerosis, the chief of these being sodium benzoate, ammonium

benzoate, hippurates, mercurial preparations, and in addition the nitrites and iodides. During the last few years, with the exception of the nitrites and iodides, he had found none of them of much use. He would confine himself to the action of the iodides. Of course, everyone here uses the iodides, but in some quarters a number of iodides have fallen into disrepute as regards their usefulness in arteriosclerosis; some observers say they are of no use at all; others again believe they are useful in some cases but not in others, and with the latter opinion he concurred.

What is the exact action of the iodides in high blood-pressure? Various opinions have been expressed. One view is that they act by reducing the viscosity of the blood; others believe that they affect the nutrition of the vessel wall; others, again, believe that iodides, through some alterative cause, have an effect on the vessel wall.

Now, first of all, iodides are useful in some cases where there is high blood-pressure, in others not at all. The cases in which iodides are useful are those in which there is high blood-pressure without any arteriosclerosis. (*Demonstration of cases of high blood-pressure treated by iodide*).

Case 1.—A man, aged 38, came complaining of headache, giddiness, and sickness for a week before admission. There was nothing wrong with the heart except accentuation of the second sound. His blood-pressure on admission was 210. During the first fortnight of his stay in hospital he had the ordinary diet, and was treated by rest in bed. The variations in pressure were shown on this slide—210-205. The patient was admitted on 9th March; iodide was given on the 27th, and at once there was a fall in the pressure as the result. On the 30th March, it was 190; on 1st April, 170; on 2nd April, 130 mm.—practically normal for this man. In this case there was no sclerosis and therefore no reason why the pressure should not go back to the normal.

An example like this gives an indication of what is the true action of the iodides. He believed that iodides act purely and simply as nitrites do. Iodide is a true vasodilator. If one substituted minutes for days in this chart then one would get exactly the same chart with a nitrite.

Now, on 16th April the iodide was stopped—one was anxious to know whether the effect was permanent or not, and desirous of making up one's mind whether the action was really due to the iodide. This other chart will show the result. On 18th April the pressure

had begun to rise; one knew that the pressure in this case must rise, because the action of the iodide is finished at the end of three days, it is out of the system. On 23rd April the pressure had risen to 197; on the 28th it was back to the original figure. Again the patient was put on iodide, this time sodium iodide, 15 grains, instead of 10 grains of potassium iodide—15 grains of sodium iodide equal 10 grains of potassium iodide; it is the amount of iodide in the preparation that must be considered. With the sodium iodide the pressure fell again in six days to 130 mm.

Case 2.—Another case of the same kind in which the pressure on admission was 175. It went down to 110 mm. under potassium iodide. Again the iodide was stopped and the pressure went up to 130 mm.; when again administered the pressure became normal.

Case 3.—Represents a case of advanced arteriosclerosis with high blood-pressure. Whether this man's high blood-pressure was due to the arteriosclerosis or not it was difficult to say. Pressure, 205-40 mm. Iodide of potassium and iodide of sodium were given with no effect at all.

Case 4.—Another case with a pressure of 225 mm. Iodide of potassium was administered and the pressure fell to 180; it remained there and no further reduction could be effected. In some cases you get a partial reduction and this was an example of that.

In treating cases with iodide one is recommended to give small doses repeated for a long time; Dr Matthew could not agree with that at all. In order to produce a beneficial effect with iodide of potassium it is necessary to begin with a minimum dose of 10 grains a day, and rapidly increase it every two or three days.

A number of organic iodides have been recommended, but these contain a very small quantity of iodide—equal to about 2 grains of potassium iodide—and he still recommended the use of the inorganic iodides.

Dr Church remarked on the great usefulness of the blood-pressure recording instrument, and advocated that it should be found in every general practitioner's consulting room. It was important in giving medicines where the blood-pressure was high not to give a remedy which would increase it, and where low a remedy that would lessen it. He had found that the administration of anything which

would increase the blood-pressure where a patient was suffering from sleeplessness due to high pressure was harmful, whereas something which reduced the pressure generally induced sleep.

The time at which the blood-pressure was taken was also an important point ; the pressure varied greatly in accordance with the time. For instance, the pressure after breakfast would be found to be 15 or 20 mm. more than that before breakfast ; after a smoke, 20 or 30 mm. more than before the smoke.

With regard to iodide of potassium, it was no doubt very helpful, but its use might be greatly increased by the help of a hot bath. He had seen a hot bath bring down the blood-pressure 25 or 30 mm. It was wonderful what a hot bath could do in lowering pressure and adding to the comfort of the patient.

As regards children, it might be that the blood-pressure affected the physical power of the child and would be a guide of what the child was able for both physically and mentally. He had tested children and found that the physical, and particularly the mental, capacity was in keeping with the amount of arterial pressure.

The *President* said he was sure they would all agree with him that they had had a very good debate. The subject was an interesting one, and it was impossible for Dr Gibson to go into it as fully as he (Dr Bramwell) could have wished, but he thought Dr Gibson had not raised many of the points that were debatable. The points in connection with the subject which required elucidation seemed to him to be, firstly, the influence which thickening or spasmodic contraction of the brachial artery had in the estimation of the blood-pressure by the Riva Rocci instrument ; secondly, in which part of the peripheral circulation was the seat of the resistance in cases of high blood-pressure—whether in the arteries, arterioles, capillaries, or larger vessels as Dr Russell supposed ; and, thirdly, was what the condition of the arterioles and capillaries in cases of high blood-pressure ?

As regards the condition of the arterial wall, he thought Dr Gibson must have modified his views ; he was under the impression that Dr Gibson had thought that the condition of the arterial wall was of no importance with regard to the reading of the blood-pressure. At all events some people had taken up that view. That position he never could understand ; he was in the habit of illustrating it by saying to his students that, if one takes a lead pipe and an india-rubber pipe and tries to compress them

one finds, of course, the greatest difference between the two; that, of course, is an exaggerated example, but it shows that the condition of the wall of the brachial artery must make some difference—how much difference is the point. Dr Elliot Dickson had found that in many cases there was thickening of the arterial coats and no increase of the blood-pressure, and from this it might be argued that the condition of the arterial coats was unimportant, but this seemed to him (Dr Bramwell) to be an illustration of one of the fallacies with regard to the reading of Martin's modification of the Riva Rocci instrument; many inches of the brachial artery are compressed by the broad band; the artery is felt to be thickened, and it is concluded because of the fact that the radial or brachial artery is thickened that absolutely the whole of the brachial artery is thickened. In many of these cases in which the brachial artery is thickened, parts of the artery are elastic and not thickened; that perhaps explains the fact that where the brachial artery is obviously thickened there may be no great increase in the blood-pressure. Very often in taking a blood-pressure observation the force of the radial pulse is found to increase for a time after the compression is applied. That is clearly explained by the increase in the peripheral resistance due to the compression of the veins—everybody no doubt has noticed that.

He agreed with Dr Gibson that in cases of polycythæmia the blood-pressure was increased; in some cases of this kind which he had observed the spleen was not enlarged.

He had difficulty in accepting Dr Russell's argument that spasm of the brachial artery is the cause of the increased blood-pressure; it seemed much more likely to be spasm of the arterioles than of the brachial artery. Of course it must be admitted that the brachial and other arteries might be thrown into spasm, but the resistance in the arterioles seemed to him to be of far greater importance.

He was surprised that nothing had been said about the capillaries; that was a debatable point.

One of the most important points seemed to be the relationship of increased pressure to arteriosclerosis. The terms arteriosclerosis, atheroma, and granular kidney, of course, include a number of different conditions. In old people there is often arteriosclerosis and atheroma with one form of granular kidney; in these cases there may be no increase of the blood-pressure. That is an entirely different condition from the arteriosclerosis with typical chronic

interstitial nephritis, sometimes seen in quite young people. The relationship of cases of high blood-pressure with arteriosclerosis where there is no obvious kidney disease to these cases was an interesting and debatable point.

A great deal of confusion existed as regards terms. Not only were thickened vessels seen in kidney disease in old people, but in syphilitics and in many other conditions. It would be well to define what was meant in individual cases by the different terms.

He entirely agreed with Dr Gibson as to the blood-pressure and the condition of the left ventricle in amyloid disease of the kidney as compared with chronic interstitial nephritis; as Dr Shennan said, pure amyloid disease is very rarely seen, it is usually associated with interestitial or parenchymatous disease; but there is no doubt that in amyloid disease the blood-pressure is low, and in chronic interstitial nephritis the pressure is high. That is a point of great diagnostic importance.

One might be tempted to say much more on this interesting subject, but at this late hour he would call on Dr Gibson to reply, and conclude by thanking him for bringing this subject before the Society.

Dr G. A. Gibson thanked the President for his graceful remarks, also those who had taken part in the discussion and those who had listened. The success of the discussion must be very gratifying to the Council of the Society.

With regard to what the President had said, he did not think he had ever in his life discounted the condition of the arterial wall, although he had not gone as far as Dr Russell. In the work of Henderson, to which he had referred, published in the *Transactions of the Royal Society of Medicine*, there were recorded variations of 35-37 mm., but what was that in a pressure of over 300? That was his great difficulty.

With regard to what Dr Russell said, he "nailed his colours to the mast" as regards the pressure being due to the resistance of the contraction in the arterioles, possibly also the capillaries—but the latter is open to doubt.

It was a pleasure to have had the observations of Dr Rainy, who had explained a good many points very clearly indeed, especially in regard to the wall of the vessel; his statement, that the tissue, although it might be called dead, is still living, largely discounts the objections of Dr Russell.

Dr Cargill Knott tried to smooth over the breach between Dr Russell and Dr Rainy, and seemed to think they might be allowed the two conditions ; but he thought Dr Cargill Knott had argued rather from a narrow premise to sweeping general conclusions. That was the impression left on his mind, but he might have misunderstood what was said.

Dr Elliot Dickson's observations were well known to him ; he had been consulted by him some years ago on the subject. The view that the thickening of the arteries in his series of cases was entirely due to carbonic acid gas was, he thought, the correct one ; it was surprising how much carbonic acid gas there was in the Fife mines.

He was interested in hearing Dr Oliphant Nicholson's paper on the explanation of a subject in which he (Dr Gibson) had a great interest ; Dr Nicholson's work on the effect of thyroid in post-partum conditions led him to expect an interesting communication from him.

With regard to Dr Shennan and arteriosclerosis, he entirely agreed with Dr Russell's view. Arteriosclerosis is entirely different from atheroma, in fact the latter is more like endarteritis deformans in its general conformation. The President had somewhat cleared up the point as regards the condition of the kidney. Under the term, chronic interstitial nephritis, there are several varieties. One observation he would like to make, and the President would remember it well, namely, that when he (Dr Gibson) was an undergraduate they used to see a great many cases of waxy disease in this city ; now they saw comparatively few. It used to be more common in Edinburgh than in the South. (President here pointed out that in his view the lessening of the disease was due to antiseptics ; it was purely antiseptic. He agreed with Dr Gibson in the fact that waxy disease was less common now, but not with his explanation of the fact.)

He had listened with the greatest interest to Dr Matthew's observations on therapeutics. He could not help thinking, when Dr Matthew was telling of his success with the iodides, with which most of them were in harmony, that in severe spasm he had found iodide of potassium act right off just as nitrites do. No doubt it was necessary to get free iodide into the blood.

He agreed with Dr Church as regards the value of the sphygmograph in general practice.

Meeting III.—December 14, 1910

Dr Byrom Bramwell, *President, in the Chair*

This was a clinical meeting held in the Royal Infirmary.

I. Exhibition of Patients

1. *Dr Dawson Turner* showed—

(*a*) Patient, aged 5, suffering from a PIGMENTED NÆVUS OF THE LEFT OCULAR REGION; this was visible at birth, but has since been growing; it measured 5 cm. each way, and was brownish black. Has been treated by the application of 13 mg. of radium for 10 hours (130 mg. hours).

(*b*) Patient, aged 25, suffering from a PORT-WINE STAIN on right cheek, temple, and neck, of congenital origin; it measured 2 by $1\frac{1}{4}$ inches. Treated by weak preparations of radium.

2. *Dr R. Cranston Low* showed—

Case of NÆVUS PIGMENTOSUS ET PILOSUS treated by CO_2 snow.

Patient is girl aged 17, who had a large dark-brown hairy nævus occupying nearly the whole of the left cheek. Frozen all over with CO_2 five times, and now shows almost complete disappearance of the lesion except for a few hairs.

3. *Dr F. Gardiner* showed—

(*a*) Case of PITYRIASIS RUBRA PILARIS.

A. M., boy, age 19. Eruption affecting face, arms, and legs—exterior aspects. Rapid onset. Yielded to internal administration of arsenic, which is unusual. $2\frac{1}{2}$ per cent. salicylic acid vaseline applied externally.

(*b*) Case of TERTIARY SYPHILITIC ULCER OF FACE.

Mrs M., æt. 35. Duration three or four years. Three first children full time—two miscarriages later. Ulcer circinate. Scars show little or no pigment.

4. *Dr J. V. Paterson* showed—

(*a*) Case of DYSTROPHIA EPITHELIALIS CORNEÆ (Fuchs).

Man, age 48, noticed visual defect about eight years ago. On left cornea, rather below its centre, a patch of epithelium roughened and œdematous or actually raised into small vesicles. On right cornea similar but rather slighter changes. Sensibility of corneæ; much impaired. Tension of eyes normal.

(*b*) Case of very advanced ARTERIO-SCLEROTIC changes in the retinal vessels of the left eye.

The lower retinal arteries show as silvery branching lines. Many small hæmorrhages are scattered over the fundus.

5. *Dr Edwin Bramwell* showed—

BROWN-SEQUARD'S PARALYSIS due to bullet wound.

Atrophic motor paralysis of right triceps, hand muscles, and extensors and flexors of fingers. Oculo-maxillary syndrome, right side. Paresis right leg, with ankle and knee clonus and Babinski. Loss of temperature and pain sensation left side below fourth rib; defect of sense of position and bone conduction on right side.

6. *Dr Norman Walker* showed—

(*a*) Case of ACTINOMYCOSIS in a labourer aged 31.

Was working among cattle and often chewed straw—Ray fungus found on September 26th. Subsequent examination negative.

(*b*) ? MULTIPLE SARCOMA OF THE SKIN.

Patient, aged 30, is a vanman. Began with vague illness; then an attack of "rheumatism" followed by one of "pleurisy." Earliest lesion on face—when seen were extensive on scalp, face, trunk, and limbs—Epitrochlear glands hard and enlarged. Some improvement under iodide.

7. *Dr G. A. Gibson* showed—

(*a*) PARTIAL ACROMEGALY, with evidence of excessive growth of the cranial bones and enlargement of sinuses, as well as of the facial bones, but no appearance of hypertrophy elsewhere.

(*b*) PATENT ARTERIAL DUCT, with the pathognomonic thrill and murmur, beginning in the middle of ventricular systole and continued beyond the second sound, which is sometimes doubled.

8. *Professor Sir Thomas R. Fraser* showed—

Cases of BERI-BERI illustrating the œdematous and the atrophic varieties; the œdematous showing the beneficial results of treatment, the atrophic the signs and symptoms of peripheral neuritis.

9. *Dr Byrom Bramwell* showed—

(*a*) Case of OBSCURE NERVOUS DISEASE.

Dock labourer, aged 29. Fœtid discharge from ear since aged 7. Well until 21, then gradually developed difficulty in moving

right arm, right leg, left arm, left leg, facial muscles, tongue. Three attacks of difficulty in swallowing.

Family history.—Good.

Present condition.—Big, robust-looking; unable to stand or walk; vacant expression; mentally bright; inability to speak; high palate; enlargement of tongue; scoliosis; movements attended with spasmodic muscular contractions, which pass off on attempting further movements; muscles react briskly to faradism and galvanism, contractions not persistent, no polar change; deep reflexes absent; plantar flexion; organic unimpaired. Cerebro-spinal fluid normal. Sensory functions normal.

(*b*) MYXŒDEMA with ascites—Cure; Tapping; Thyirod extract.

Mrs W., aged 50. Typical myxœdema of six years' duration, following severe "floodings." Thyroid treatment; well for three years; then influenza; thyroid treatment suspended; redevelopment of myxœdema. On admission, weighed 15 st. 4 lbs; abdominal measurement 48½ inches. Thyroid treatment; 400 ounces of yellowish-green fluid withdrawn; no reaccumulation of fluid; weight reduced to 10 st. 11½ lbs; complete disappearance of myxœdematous condition.

10. *Mr Caird* presented two cases of PYLORIC GASTRECTOMY FOR CARCINOMA—

(*a*) The first case was that of a man, W. R., aged 28, who had suffered for a year from symptoms suggestive of chronic gastric ulcer, for which he had been treated in the medical house with only temporary benefit. There was no marked diminution in hydrochloric acid; after a test meal there was some residual food.

Laparotomy, on 19th November, revealed a firm mass around the pylorus, under cover of the liver. It was of "wooden" consistence as if due to chronic inflammation, was covered with adhesions, but posteriorly gave such a definite stony sensation to touch that malignant disease was recognised and excision carried out. During the main operation the large ulcer gave way in the vicinity of adhesions to the pancreas, and a considerable escape of gastric contents took place. The pancreas was also lacerated during removal of the growth. Local drainage and a tube in the pouch of Douglas was thought advisable.

(*b*) The second case, Mr A. A., aged 55, had a year's history of indigestion; during the last six weeks he had emaciated rapidly. There was a large, slightly motile mass filling the gastric area. On

operation there was found to be a large carcinoma so involving the meso-colon that the transverse colon had to be sacrificed. In order to favour repair of the end to end union of the colon an appendicectomy was performed. The pouch of Douglas was also drained.

11. *Mr Alexis Thomson* showed—

(*a*) Removal of impacted stone from the lower end of each ureter. The patient, a man aged 29, a miner, had had symptoms pointing to renal and ureteral stone for four years. The X-rays showed two small shadows placed symmetrically at the level of the floor of the bladder. The stones were removed by the vesical route.

(*b*) PERIOSTEAL SARCOMA OF HUMERUS in a boy aged 12, already shown to the Society. The musculo-spiral paralysis has been recovered from, but the lower half of the humerus became the seat of new growth and was resected on the 6th September. A portion of the right fibula was inserted in its place.

12. *Mr David Wallace* showed—

(*a*) Mrs H., 30 years of age, suffered from pain in the right lumbar region in August 1910. No urinary symptoms until September, when she had rigors, and the urine contained much pus and was alkaline. October 19th, cystoscopy showed a healthy bladder. Perinephric abscess detected 4th November. It pointed in 9th intercostal space. Opened and pus showed gram negative bacilli. *Nephrectomy*, November 29th. Localised abscess.

(*b*) Mrs C., æt. 36, complained of difficulty in swallowing for three months. Œsophagoscopy showed an annular stricture at upper end of œsophagus. Portion removed microscopically squamous epithelioma. *Operation*: excision of tumour, upper end of œsophagus fixed to muscles and skin, and œsophageal tube introduced. No enlarged lymph glands.

13. *Mr H. J. Stiles* showed—

(*a*) R. F., aged 13 years. Admitted into Chalmers Hospital, June 29, 1910. He complained of pain in the abdomen, which was attributed to a fall four weeks previously. Two days after the fall he passed a considerable quantity of dark blood-stained urine. His abdomen was said to have been large since birth, and during infancy he suffered from frequent attacks of vomiting. He is said to have had meningeal symptoms on two occasions when a child.

On examination there was found to be very marked general distension of the abdomen, with a feeling of elastic resistance all over. The whole abdomen was absolutely dull to percussion, with the exception of the left iliac and the outer portions of the left lumbar and hypochondriac regions, all of which were tympanitic. There were well marked palpable percussion waves, and palpation gave one a feeling that the abdomen was almost completely filled by one large cyst. The percussion signs were not affected by position. The circumference of the abdomen at the level of the umbilicus was 27½ ins. The liver dullness reached up to the upper border of the 4th rib. The apex beat was in the third left interspace slightly internal to the mammary line. The urine was alkaline ; its specific gravity was 1008, and it contained a trace of albumin and a considerable quantity of altered blood. The microscope showed red blood corpuscles and a few leucocytes. Urea = 1 gr. per oz.

The cyst was diagnosed to be a very large congenital hydronephrosis of the right kidney. Nephrectomy was performed by an extra-peritoneal lumbo-iliac incision. On reaching the wall of the cyst the latter was tapped and 7½ pints of dark brown fluid with a urinous odour were withdrawn. As the cyst was being emptied its wall was gradually pulled out of the wound and the peritoneum and colon were then gradually separated from the cyst until finally the pedicle was reached. The latter was broad and sheet-like. The renal artery was small, but the pedicle contained several large and very thin-walled veins. The pedicle was so broad that several ligatures were applied to it so as to include in separate portions the ureter, the artery and the large veins. The lowest portion of the sac was the most adherent. After dropping back the stump, the wound was closed with the exception of a small portion through which a roll of ivorine was introduced down to the pedicle for drainage. There was a very thin layer of kidney substance stretched over the cyst wall, and in one portion of the sac small calcareous plates were present.

The fluid from the cyst had a specific gravity of 1013, was neutral in reaction, contained a considerable number of red blood corpuscles, a few leucocytes, 2 grs. to the oz. of sugar, and 1.9 grs. to the oz. of albumin. There were no casts.

The patient made an uninterrupted recovery.

The case is a good illustration of the very large size to which a hydronephrosis may attain. Attention had evidently been directed

to the condition by the blood-stained urine, no doubt due to the injury to the cyst, the result of the fall.

Mr Stiles remarked that he had brought a similar case before the Society some few years ago. (See Vol. XXII., New Series, 1902-1903, p. 291.) In this case the cyst was excised by the transperitoneal route through a mesial incision at the level of the umbilicus. Mr Stiles had come to the conclusion that the transperitoneal method was the simpler, and, on the whole, the one to be recommended.

(*b*) J. G., male, aged 26 years. Admitted to Chalmers Hospital, October 24, 1910, as an ACUTE INFLAMMATORY CONDITION WITHIN THE ABDOMEN complicated by obstruction.

The history was that the patient had complained of pains in the joints and malaise for three weeks before admission, and that two days previously he was suddenly seized with severe pain in the right iliac and lumbar regions. His appendix had been removed a year and eight months previously. The pain had been continuous; there had been considerable nausea but no vomiting. The bowels had been very constipated, and there had been great thirst but no urinary symptoms.

On admission the patient did not look very acutely ill; the tongue was dry and thickly coated. Temperature 101.8 and pulse 120; leucocytes 26,000, with a positive glycogenic reaction.

Examination.—The abdomen was found to be slightly distended and rather tense throughout, the right rectus, however, being more rigid than the left. There was tenderness in the right iliac region and right loin. The percussion note was slightly tympanitic throughout.

Urine.—Specific gravity 1020, with a trace of albumin but no blood. Urea, 3 grs. to the oz. Microscopic examination showed numerous pus cells, a few red blood corpuscles, numerous cocci and bacilli, but no casts.

As the pain and tenderness were most marked in the region of the scar, the result of the appendectomy, the abdomen was opened in the line of the old incision. There were no adhesions about the ilio-cæcal region, and apart from the somewhat distended condition of the bowel there was nothing abnormal to be made out in the intestine. A large, deep-seated, rounded swelling was felt in the loin behind the ascending colon, the nature of which could not at first be determined. By enlarging the incision upwards and backwards and dividing the peritoneum outside the colon and separating the

latter, the swelling, which was the size of a fœtal head, was found to occupy the lumbar region and to extend upwards beneath the costal margin. On separating the retro-peritoneal tissue, it was found to be a very tense, acutely inflamed hydronephrosis. This was then tapped, and 10 ozs. of dark, blood-stained, semi-purulent fluid mixed with flakes of lymph was drawn off. As the peritoneal cavity had been freely opened, and as it was not possible to drain the cyst without infecting the peritoneum, it was deemed advisable to excise the kidney. This was found to be a difficult matter on account of (1) the distended condition of the intestine, and (2) on account of the extensive adhesions of the upper part of the cyst to the adjacent structures. After still further enlarging the wound upwards and backwards, it was found necessary to make a second incision upwards and inwards at right angles to it. The lower pole of the cyst was then freed sufficiently to expose the ureter and then the vessels forming the renal pedicle. Having clamped and divided these the upper pole of the kidney was then separated from the liver and posterior abdominal wall by introducing the whole hand and separating it by blunt dissection with the fingers. There was considerable bleeding during this part of the operation, the bleeding, however, being due to numerous veins in the adhesions. The important point in the operation was the securing of the pedicle before dealing with the upper pole of the kidney. After introducing one drain deeply into the lumbar region and a second down into the pelvis, the rest of the wound was closed.

The patient made an uninterrupted recovery, and left the hospital towards the end of the fifth week.

The kidney showed intense congestion of the atrophied cortex and medulla, the latter being encroached upon by the cystic dilatation of the calices which contained large greyish sloughs. The pelvis was greatly dilated and lined by an intensely congested white-looking mucous membrane. The capsule was thickened.

Cultures from the fluid contained the staphylococcus albus, a bacillus of the proteus type, and a gram positive saprophyte.

14. *Mr George Chiene* showed—

A patient, Mrs J. L., aged 24, operated on three months previously for RUPTURED OVARIAN ECTOPIC GESTATION.

Three days before admission to hospital patient complained of abdominal pains, especially on the right side. The next day, after taking castor oil, she felt better and did her house work. The

following day there was no further change, but on the day of admission to hospital, severe abdominal pain, which made her cry out and collapse on the floor, came on about one o'clock. Her doctor was called in and gave her an enema with good result, but the pain continued, so he sent her to the Infirmary the same evening. On closely questioning her she admitted that she had had a faint turn at the time of the severe attack of pain, also several other faint turns during the afternoon. Previous health very good. Menstrual history: period every four weeks, four days' duration, had not missed a period and expected to be unwell three days later. Last period perfectly normal. Seven healthy children, last child two years old.

State on admission.—Patient looked ill and pale, said she felt very ill and complained of great pain. Temperature, 99.6; pulse, 120. The abdomen was slightly distended, but moved fairly well on respiration. On palpation there was marked general abdominal tenderness, more pronounced in right iliac fossa and above the pubis. Vaginal examination caused very great pain, and on rectal examination a fulness, tender to the touch, was felt in the pouch of Douglas.

The patient was sent to hospital as a probable case of acute appendicitis, but on examination ectopic gestation was suspected. The fact that the patient had not missed a period was, however, misleading.

The abdomen was opened through the right rectus muscle and was found to contain a large quantity of blood and blood clot. The uterus and tubes were examined and found normal, but the right ovary showed a rupture, about the size and colour of a ripe cherry, at the end nearest the uterus. There were no adhesions, and the corresponding tube was normal. The ovary was removed, the clots cleared out, and the abdomen filled with normal saline and closed.

For two days her condition gave rise to some anxiety, but after that she rapidly recovered.

There was a possibility of conception having occurred twice since the last menstrual period, once two days after and again twelve days later.

The ovary was preserved whole, and serial sections are being prepared. Those so far examined show typical chorionic villi. A water-colour drawing, showing the appearance of the ovary after removal, was exhibited.

15. *Mr J. W. Struthers* showed—

(*a*) Case after nephrotomy for ESSENTIAL HÆMATURIA.

Mrs A., æt. 35. History of hæmaturia for 2 years, variable in amount, but never completely absent—Found to be bleeding from left kidney only. Kidney exposed, split open, and nothing found; stitched up with through-and-through catgut sutures. Gradual complete cessation of hæmorrhage.

(*b*) Case after operation for ACUTE OBSTRUCTION due to a band, and also to tuberculosis of termination of ileum. Band divided and lateral anastomosis established between ileum above disease and ascending colon; diseased gut not resected. Patient now apparently perfectly well.

16. *Mr Henry Wade* showed—

(*a*) Patient three months after excision of the diaphysis of the ulna for ACUTE INFECTIVE OSTEOMYELITIS.

D. R., boy, aged 4½ years, developed acute infective osteomyelitis which was treated by passive congestion and incision, followed by excision of the ulna 21 days after onset of disease. Primary union. Complete osseous regeneration.

(*b*) Patient one month after nephropexy for MOVABLE KIDNEY.

Boy, aged 4 years, operated on for an extremely mobile tumour in the right iliac fossa, which was found to be a movable kidney.

17. *Mr D. P. D. Wilkie* showed—

(*a*) Patient (male, æt. 19), after operation for PERFORATED DUODENAL ULCER, to illustrate benefit from intraperitoneal administration of oxygen.

(*b*) Patient (male, æt. 36), after implantation of omentum in abdominal wall for HEPATIC CIRRHOSIS WITH ASCITES.

Meeting IV.—January 11, 1911

Dr BYROM BRAMWELL, *President, in the Chair*

I. ELECTION OF MEMBER

Harry Moss Traquair, M.D., D.P.H., F.R.C.S. Ed., 30 Walker Street, was elected an Ordinary Member of the Society.

II. EXHIBITION OF PATIENTS

Mr Charles W. Cathcart showed—

(*a*) Girl showing the advantage of resuture of the facial nerve when divided during removal of a PAROTID TUMOUR. The tumour

was deep-seated, and the res anserina was stretched over it. The injury was not recognised till recovery from the anæsthesia. Next day the wound was reopened and the surface searched with electrodes for the distal ends of the divided nerves. When found, these were united to the proximal ends, and recovery has resulted.

(*b*) Man with large FISTULOUS OPENING just above hyoid bone, resulting from attempted suicide from cut throat. It is possible to demonstrate in this case some interesting points as to the mode of formation of the vowel-sounds.

III. Exhibition of Specimen

Mr J. M. Cotterill showed—

Unique specimen of diverticulum of the cæcum, containing a very large concretion.

IV. Demonstration

Dr A. D. Webster gave a Demonstration on Exercises.

By wall diagrams the general effects of exercises were illustrated during adult life. Also, the particular effects during adolescence against stoop, spinal curvature and flat-foot. These among lesser errors were shown to be capable of prevention and correction by appropriate exercises. He pointed out the importance of the principles being carefully adapted to each case by the practitioner, and also the results of good work and of neglect, as well as of overuse or wrong use, of exercise.

V. Original Communication

THE PRESENT POSITION OF THE TREATMENT OF PLAGUE

By J. C. Thomson, M.A., M.D., D.P.H., D.T.M., & H. Camb.

Mr President,—The question of the treatment of plague is fortunately one of only academic interest in Edinburgh. A few weeks ago, however, considerable anxiety prevailed throughout the country when the investigations that followed the occurrence of a number of cases of pneumonic plague in the county of Suffolk, revealed the presence of an extensive epizootic of the disease among the rats not only of that county, but of the neighbouring counties as well. It was at that juncture that it was suggested that this Society might profitably discuss the present position of the treatment of plague; and that, having had much to do with the disease in Hong-Kong, since it became endemic there in 1894, I might prepare a brief

review of the subject. In the time available it is, of course, possible to submit only the barest outline of many matters one might usefully have described in detail.

Before discussing some of the more important of the special methods that have been advocated for the treatment of plague, by means of antiseptic drugs, serum-therapy and organo-therapy, none of which can claim much success, I propose to emphasize certain points in reference to the general management of this disease.

General Management of a case of Plague—*Initial Precautions.*—Pneumonic cases, fortunately few in most epidemics, are virulently infectious through the emanations from the lungs, and should be dealt with in hospitals where due precautions against spread of the disease can be adequately provided; but bubonic and septicæmic cases may be safely treated in the wards of ordinary hospitals, or in their own homes, provided that measures against rats and their fleas be taken. The Second Indian Plague Commission (1905-1907) proved conclusively that the rat-flea is the principal, and probably the only, transmitter of plague, other than the pneumonic form. Soon after the publication of its report the outlying special hospitals in Hong-Kong, for instance, were abandoned to other uses; and all plague cases are now treated in the general hospitals, or in cottage hospitals scattered throughout the city, and so placed as to obviate the necessity for conveyance of the sick to any great distance. Previous experience in that colony had already shown that the members of the staff of a plague hospital run practically no personal risks.

If a case is to be treated at home, rats should be hunted out, and disinfection with special reference to destruction of fleas must be done,—not after recovery, but as soon as the diagnosis is declared.

Combined methods should be employed for the local extermination of possibly infected rats:—

Traps should be set, with baits varying in their character, form, and appearance from day to day.

Poisoning may be done by means of a paste containing 2.5 per cent. of phosphorus; or by barium carbonate,[1] made up as a dough with meal in the proportion of one part to four, or spread on fish or moist toasted bread.

Birdlime, spread on boards and baited like the traps, is very effective; and Danysz's virus, which kills the rat by inducing a

[1] *British Medical Journal*, Dec. 17th, 1910.

specific septicæmia, although it is uncertain in its action, should also be used, if available, spread on bread and exposed near the rat-holes.

Clothing, bedding and the like may be disinfected by steam; but it is to be noted that the ordinary gaseous and chemical disinfectants for infected rooms are useless against fleas. These insects may, however, be readily destroyed by the application to bedsteads, walls, floors, etc., of a mixture of petrol and cyllin, equal parts, diluted with water for use. This is a combined pulicide and germicide; and in a dilution of one part of the mixture to 200 parts of water has been proved, experimentally and practically, efficient and reliable for the destruction of both fleas and plague bacilli.

Contacts.—Contacts, *i.e.* persons who may presumably have been exposed to the bites of fleas in the circumstances where infection of the patient occurred, should be inoculated with Haffkine's prophylactic, which not only protects the healthy for a period of several months, but aborts the incubating disease. This is a broth culture of the plague bacillus, killed by heat, and containing the dead bodies of the bacilli and the products of their growth, with a minute quantity of carbolic acid added as a preservative.

Other prophylactics have been prepared by Lustig and Galleotti, Terni and Bandi, Strong and Kolle, and Klein; but Haffkine's fluid has maintained its reputation as the safest and most efficient.

Disinfection during the illness.—In reference to the question of arrangements for general disinfection during the illness, excepting in cases of the pneumonic type,—which, as I have mentioned, are comparatively rare, but virulently infective from person to person, and therefore requiring the most complete measures possible in this respect,—the arrangements for disinfection in plague may perhaps best be described as approximately the same as for enteric fever. It is abundantly proved that the rat, which is the principal source of danger to human beings, is rarely infected through the alimentary canal; but it certainly may be so infected, and since the fœces, urine, sputum, etc., of a plague patient frequently contain plague bacilli, adequate means for their disinfection should be taken before they are put into the drains. Carbolic acid, 1 in 20; cyllin, 1 in 200; or, corrosive sublimate, 1 in 500 in an acid solution, may be used.

Nursing.—Careful nursing of a case of plague is of the first importance. On account of the marked tendency to heart failure in this disease the recumbent position is essential so long as acute

symptoms last, and in delirious patients, who are often wildly resistive, has to be maintained by mechanical means not infrequently. The patient's mouth should be cleansed systematically with a solution of boric acid or the glycerine of the same, and his body sponged daily with a dilute antiseptic, such as cyllin in tepid water.

Retention of urine is frequent, and close attention to the state of the bladder is necessary. Doors and windows should stand open day and night, and bedclothes should be of the lightest description. Nurses should be warned of the possibility of infection through skin wounds, and even the slightest abrasion of the hands should be carefully closed with collodion. All unnecessary visitors should be excluded.

Food.—Food should be of a bland character, liquid, and given in small quantity frequently; but after acute symptoms subside the diet may be rapidly improved. Cold water should be freely at disposal between the feeds, and should be frequently forced on acutely ill or delirious patients.

Stimulants.—The use of cardiac stimulants should be a routine practice in all cases of plague from the very first. Great prostration occurs as a rule within a few hours from the commencement of the illness, and in the earliest stages the debilitated heart needs support. There is great variety in the drugs that may be used, and considerable difference of opinion as to the value of, or necessity for, the use of alcohol. My own practice has been to give, in an ordinary moderate case, brandy, 4 oz., spread over the twenty-four hours in ½ oz. doses; and, in addition, I administered strychnine hypodermically, four-hourly, or a mixture containing strychnine and digitalis by the mouth. Other stimulants one called into requisition as circumstances required. Choksy of Bombay speaks favourably of supra-renal gland, which he used in the form of Oppenheimer's renoglandin, as a remedy for heart failure in plague.[1]

Symptomatic Treatment.—The further treatment of plague is chiefly symptomatic; attempts to find a specific remedy, to which I am about to refer, have been made, and some success has been claimed, but a plague specific is still in the future.

If there be constipation at the beginning of the illness, a purge of calomel, followed by a saline, is useful; later, it is better not to give any strong purgative, but to open the bowels by means of enemata.

Diarrhœa occasionally occurs, when the discharges are found to

[1] *Indian Medical Gazette*, April 1905; Feby. 1907.

be loaded with plague bacilli ; and intestinal antiseptics are called for, with increased solicitude for the maintenance of the general strength, meat preparations being meanwhile discontinued, and milk diluted with lime water or peptonized ; while in severe cases opiates should be used.

For the reduction of high temperature in plague, the depressant antipyretics, such as antipyrin and phenacetin, should never be employed. Ice-cold applications should be used ; but the risks of collapse must in this, as in every other detail of the treatment of plague, be kept in view continuously, and as soon as the rectal temperature comes down to 102°F. the cold should be removed, lest the downward tendency continuing after removal should lead to disaster.

Cold applied locally is also of value for headache and the control of delirium. Headache may yield to caffeine citrate. For delirium and insomnia resort has often to be made to hypnotics ; and probably the most generally useful is morphia sulph., gr. $\frac{1}{4}$-$\frac{1}{2}$, or hyoscine hydrobromid., gr. $\frac{1}{100}$, given hypodermically, alone or with such drugs as chloral and potassium bromide, trional, etc., by the mouth.

Calcium chloride or adrenalin may be used to control hæmorrhage.

Pneumonic Plague.—No special treatment for the pneumonic form of the disease is indicated, other than the general measures already described ; but on account of its intense infectivity, rigid isolation is necessary, and nurses should be specially warned as to careful personal prophylaxis.

The Bubo.—The treatment of the plague bubo has been the subject of much discussion, but the consensus of opinion is against excision of it. A large proportion of the buboes of patients who recover resolve without suppuration, and it is not possible to say beforehand what will be the termination of any particular bubo. My treatment has been entirely conservative :—green extract of belladonna and glycerine, in equal proportions, were applied freely on lint in the early stage ; warm poultices were used when suppuration was seen to be inevitable ; a simple incision, followed by scraping with a blunt spoon, was done when suppuration was well established ; and the resulting wound was treated on ordinary surgical principles. The application of an ice-bag to the early bubo relieves pain and tenderness, and may be used in addition to the belladonna.

Injections into the bubo of carbolic acid, iodine, corrosive sublimate, cyllin and other antiseptics have been advocated, but there is no evidence that substantial benefit has resulted from any of them.

The Point of Inoculation.—The point of inoculation of plague should always be looked for, and, if discovered, it should be attacked vigorously. Unfortunately, it is rarely to be found. In experimental plague, while the point of inoculation is marked by œdema in ordinary laboratory animals, there is usually no apparent lesion in monkeys; and this accords with what occurs in human plague. But occasionally there may be seen on a limb, below the bubo, a papula with an inflamed base and a clear vesicle at the top, the contents of the vesicle swarming with plague bacilli in pure culture. The interpretation seems to be that the advance of the bacilli has for some reason been arrested, and that the bacilli are proliferating locally, pouring their toxines, only or chiefly, into the circulation. In any case, destruction of the papula is usually followed by abortion of the illness and a rapid recovery. The contents of the vesicle should be received on a swab moistened with strong carbolic lotion; the papula should then be transfixed, and a deep crucial incision made; and finally, the tissues should be deeply cauterized with pure carbolic acid applied by means of a piece of pointed wood.

Ulcers and Carbuncles.—The indolent ulcers that sometimes result from the buboes, and the gangrenous skin areas, so-called carbuncles, occasionally met with, are only manifestations of the general depression, and their treatment is on general principles.

Convalescence.—Convalescence from plague is usually slow, and the profound enfeeblement frequently resulting from the illness, with the mental and other sequelæ that sometimes occur, have to be dealt with as individual cases demand.

Specific Methods for the Treatment of Plague.—It remains to discuss a number of efforts made to find a specific remedy for plague, in the directions of antiseptic drugs, serum-therapy and organo-therapy.

Antiseptic Drugs.—Attempts have been made to introduce carbolic acid into the tissues in sufficient quantity to delay the proliferation of the plague bacillus, and allow the natural reaction against the organism time to assert itself.

In 1901 I used this drug in doses of 80 grains a day in a series of 204 cases, but, while the Hong-Kong plague hospital

mortality in that year was 76.5 per cent., slightly less than the average, I did not think it had been much affected by the treatment.

In 1902 I used serum treatment, with negative results, to which I shall presently refer; but in 1903 I returned to my experiments with carbolic acid. I determined to push the remedy to the utmost, and, while watching carefully against appearances of poisoning, to give the drug in massive doses so long as plague bacilli could be detected in the blood or serum from the bubo. I gave 144 grains of pure carbolic acid daily, divided into two-hourly doses of 12 grains each, administered in a mixture with syrup of orange and chloroform water.

Carbolic acid, thus administered, was taken in large amount by some of the cases ere the prescribed condition was satisfied. To one patient, for instance, a severe septic case, non-bubonic, in a European, in which violent delirium required the use of shackles for nearly a week almost continuously day and night, over 2,500 grains of pure carbolic acid were given before the blood was free from plague bacilli: an excellent recovery was made, and the patient is now a strong healthy man, still in the Far East.

Carboluria, which is probably due more to impurities in the acid than to the drug itself, rarely occurred in course of this series of experiments; where it did occur, the omission of one or two doses was usually sufficient to clear the urine and allow resumption of the remedy. In certain cases dyspeptic symptoms supervened, but in these greater dilution of the mixture with water was sufficient to overcome this obstacle to its consumption.

The drug was thus employed over a series of 143 cases, and the mortality was only 36.4 per cent. Unfortunately, in the earlier part of the epidemic of that year I had been testing the curative value of the antiseptic oil of cinnamon, which proved useless; and the results with carbolic acid were obtained in the later part of the epidemic, when there is a greater natural tendency to recovery, the disease being invariably more virulent early in the epidemic; so for this circumstance due allowance had to be made.

Further experience in Hong-Kong has confirmed my belief that carbolic acid, while in no sense a specific remedy, is of undoubted value in the treatment of plague when given in such doses as to allow its sufficient concentration in the blood to exert an inhibitory influence on the proliferation of the plague bacillus.

Other observers, however, have reported less favourably on the

results of its use in India, etc.; and trials made with other antiseptics, such as formic aldehyde, injected intravenously; cyllin, injected intravenously; oil of cinnamon by mouth, etc., have been even more disappointing.

Serum-Therapy.—The position as regards the serum treatment of plague is pretty much that described in reference to carbolic acid and other antiseptics. Most unequal results have been obtained by different observers, among different races and in different epidemics. In comparing reports of results, by any or all the methods of treatment, it is important to note that European races show a much larger proportion of recoveries, irrespective of treatment.

In 1902 I treated in Hong-Kong a series of 94 cases with a serum supplied to me in monthly instalments by Calmette of the Pasteur Institute in Paris, using the method of administration and the quantity recommended by him. The average initial dose was 40 c.c. intravenously and 20 cc. subcutaneously on admission, and then 20 c.c. subcutaneously on each subsequent day while fever lasted. The hospital mortality was 85 per cent., higher than in any previous year; and I concluded that the serum was of no value, though deterioration through lapse of time and through exposure to hot temperatures during transit from Paris probably contributed to this result.

Serum treatment has been used in Hong-Kong repeatedly in the years that have followed, but with no better results.

On the other hand, in Oporto, 140 cases were treated, with a mortality of only 14.78 per cent.; and in Brisbane 56 persons were treated, with a mortality of 44.6 per cent.; 39, with a mortality of 17.2 per cent.; and 65, with a mortality of 13.8 per cent. In reference to all these, the European nationality is to be borne in mind.

As regards India, N. H. Choksy, whose opportunities for the observation of the treatment of plague in the Maratha Plague Hospital at Bombay are very large, has recently reported on the subject.[1] He tested the value of the Yersin-Roux serum supplied by the Pasteur Institute in Paris. He rejected cases already septicæmic and unlikely to benefit by any treatment, as also convalescent and semi-convalescent cases, and patients who had passed the fifth day of the illness. Observation was thus limited to cases of acute illness within the first five days from the onset. Alternate

[1] *British Med. Journal*, May 30th, 1908. *Transactions of the Bombay Med. Congress*, 1909.

cases were treated with serum, the others being observed as controls. Results were as follows :—

200 control cases, mortality 74 per cent.
200 serum cases, mortality 63.5 per cent.

Difference in favour of serum treatment, 10.5 per cent.

Of 468 cases treated in private practice with serum, and in which there was a larger proportion of cases treated on the first or second day of the illness, with better social circumstances, the mortality was only 39.9 per cent.

Other serums were tested, *e.g.* Lustig's, Terni's, Cruz's, Kitasato's, etc., but results with all others were less favourable.

Indian experience generally seems to indicate that case-mortality is but little affected by the use of any serum yet prepared. But the course of the illness is favourably influenced, especially if serum be used on the first day of the disease. There is a reduction of temperature, a lower rate of pulse and respiration, less delirium and restlessness, more tendency to resolution of the bubo, less general discomfort, and a more rapid convalescence.

Intravenous injection is probably most efficacious, with at the same time subcutaneous injection into the lymphatic area draining towards the bubo ; and instead of the doses at first recommended 200 c.c. or 300 c.c. should be given.

Organo-Therapy.—Another method for the treatment of plague, the use of adrenalin chloride, is described by E. N. Thornton from his experience in South Africa.[1] This drug was advocated as a special remedy in the *Calcutta Practitioner* in 1904, by Rai Bahadur Kailaschandra Bose ; and we have already referred to Choksy's use of renoglandin as a measure for the arrest of heart failure in plague ; but the suggestion from South Africa is claimed as independent.

Thornton was led to the use of this powerful cardiac stimulant and constrictor of the peripheral blood-vessels by a consideration of the extreme congestion of all the internal organs so characteristic of plague, and in particular by the marked degeneration that occurs in the supra-renal glands, loss of whose function, he surmised, might account for some at least of the phenomena of this disease.

He used Parke Davis and Co.'s 1 : 1000 solution of adrenalin chloride in doses of m.xxx., combined with tincture of strophanthus, m.x., by mouth, four-hourly for the first three days, and then thrice

[1] *Lancet*, April 9th, 1910.

daily during a further period of fourteen days. Later experience led him to give the drug at first subcutaneously or intravenously, and in part into the neighbourhood of the bubo.

The mortality among 50 cases thus treated was 26 per cent.; while in 936 cases before the use of adrenalin the mortality had been 37.4 per cent.

The blood-pressure was raised, the heart stimulated, collapse averted, and less extravasation occurred around the bubo; but there was no effect on the delirium or the insomnia.

There is thus an apparent advantage; but the number of cases is insufficient to justify more than an opinion that the remedy is worthy of more extended trials.

Summing up in a word, Mr President, the present position of the treatment of plague is exceedingly unsatisfactory; in spite of earnest work devoted to it in all parts of the world during the recent pandemic, our ability to deal clinically with this very fatal and perplexing disease has advanced but little since the question first in modern times became urgent in the Hong-Kong epidemic of 1894.

We have still to place chief reliance on careful nursing and general management as for an acute asthenic fever. For the rest, where fresh serum is available, I think it should be used, together with adrenalin, for advantages it certainly seems to possess; where serum absolutely fresh is not to be had, I would still pin my faith to carbolic acid, and give adrenalin along with it.

And meanwhile we live in the pious hope that sooner or later a serum may yet be evolved that will prove really efficient as a plague remedy.

Meeting V.—February 1, 1911

Dr Byrom Bramwell, *President, in the Chair*

I. Exhibition of Patients.

1. *Professor Caird* showed—

(*a*) Retroperitoneal dermoid.

Patient, R. S., female, æt. 45, complained for two months of a swelling in the upper part of the abdomen. Abdomen explored, 20th December 1910. A retroperitoneal tumour found, situated to the left of the mid-line, behind the pancreas, and in front of the

lower pole of the left kidney. The tumour was adherent to and was obstructing the left ureter. The latter was divided and subsequently repaired by end-to-end anastomosis.

(*b*) CARCINOMA OF THE RECTUM removed by the Abdominosacral Method.

A. R., female, æt. 57, symptoms of rectal carcinoma for four months. Rectal examination revealed a large tumour encircling the bowel just within reach of the finger. Operation, 20th December 1910. Abdomen explored, tumour felt at the floor of the pouch of Douglas, and peritoneum round it divided. Parasacral incision, division of lower end of sacrum, delivery of tumour through sacral wound, resection, end-to-end suture.

2. *Mr Alexis Thomson* showed—

Boy, aged 13, run over by motor car, 27th October 1910, admitted R.I.E. unconscious. Some blood in the vomit and in the urine; apparent complete recovery; readmitted 29th November 1910 with sickness, and swollen, tense abdomen, due to accumulation in lesser sac. The following day abdomen slack; fluid had escaped into greater sac. Incision and drainage of both sacs. Discharged and readmitted on the 30th January 1911, with tense accumulation in lesser sac.

II. ORIGINAL COMMUNICATIONS

1. A LOCAL EPIDEMIC OF ACUTE ANTERIOR POLIOMYELITIS

By D. W. CURRIE, M.D., B.Sc., D.P.H., and
EDWIN BRAMWELL, M.B., F.R.C.P. Edin. and Lond.

INTRODUCTORY

No disease has occupied such a prominent place in neurological literature during the past two or three years as has acute anterior poliomyelitis. The reason is to be found in the numerous epidemics which have recently occurred in various parts of the world. It is true that the disease appears to have been met with in "epidemic" form in Scandinavia for more than two decades, but in other parts of Europe and in the United States epidemics were almost unknown prior to the commencement of the present century.

We propose in this paper, *firstly*, to describe a localised

"epidemic" of acute anterior poliomyelitis which has occurred recently in the practice of one of us (D. W. C.); and, *secondly*, to bring forward evidence which seems to justify the assertion that the disease has been more prevalent than usual in many parts of the country during the autumn of 1910.

The "epidemic" here recorded is, we venture to think, of very exceptional interest and importance from the suggestive data it affords in relation to the incubation period of the disease.

The Harvieston Epidemic

The five cases reported in this paper all occurred in a farmstead consisting of four houses situated on a private estate some distance from the main thoroughfare, and two miles from the town of Tillicoultry (pop. 3,600), in the County of Clackmannanshire.

The four houses may be conveniently designated A, B, C, and D. House A is occupied by the factor or land-steward, the three adjacent cottages by farm employees and their families.

The A family consists of two children (A. A., aged 5, and B. A., aged $2\frac{1}{2}$).

The B family consists of four children (A. B., aged $7\frac{1}{2}$; B. B., aged $5\frac{1}{2}$; C. B., aged 4; and D. B., aged 7 months).

The C family consists of four children (all above 8 years of age).

The D family consists of two children (aged 2 years and a few months respectively).

Five of the children in these four houses were attacked—(A. A., B. A., A. B., B. B., and D. B.).

Record of Cases

The following is a brief record of the history and symptoms of the five cases in the order in which they were taken ill.

Case 1.—B. B., aged 5 years and 6 months, came home from school on the 12*th* and 13th of September complaining of headache, which he referred to the forehead. He slept well each night, but on the 14th, as he still complained of headache and was feverish, his mother kept him in bed. During the day his mother states that he complained of pain in the back of the neck and in the legs, while she noticed that he "held his head back." He was drowsy and heavy all day. For the next forty-eight hours he was feverish and restless, but on the 16th the fever had quite disappeared. The bowels were constipated. There was no vomiting. On the 19th,

when he was allowed to get up, he complained that his legs felt very tired, and it was observed that his head, which he seemed to have difficulty in holding up, was inclined to the right side. In a few days he appeared to be quite well and returned to school on September 26th. He walked to school and back, a distance of two miles each way.

Case 2.—A. B., aged 7 years and 4 months, a sister of the last patient, complained of headache on the morning of September 16*th*. She went to school as usual that day, but when she returned in the afternoon she said that the pain in her head was very severe and that the legs felt so tired and sore that she had had difficulty in getting home. She was feverish and did not sleep that night. The fever continued for two or three days. Her mother states that her neck was stiff and she held it far back " just like Archie " (B. B.). The pain in the legs was referred chiefly to the right hip and to both knees. Pain on passive movement of the limbs was such a striking feature that the condition was at first suggestive of acute rheumatism, notwithstanding the absence of any articular swelling. She did not vomit. The bowels were somewhat constipated. The pain in the legs was severe for a week, and it was only on the third or fourth day of her illness that weakness of the lower limbs, more particularly the right, was observed. For two or three days she seems to have had a little difficulty in passing water. Improvement has been slow.

When examined on October 31st, it was found that there was complete paralysis of the right lower limb, with the exception of a slight degree of movement of flexion and adduction at the right hip. On the left side all the movements were fairly satisfactorily carried out against resistance, although flexion at the hip was relatively weaker than the others. There was at this time no observable weakness of the trunk muscles. The upper limbs were in all respects normal, as were the cranial nerves. The paralysed and paretic muscles showed no wasting as compared with those of the left lower limb, unless in the case of the thigh which was very slightly smaller than its fellow. The affected muscles were quite flaccid. The foot on the right side could be brought to within a few inches of the face when the extended leg was flexed on the trunk, while that on the left side could only be brought to about the vertical. The muscles of both lower limbs were slightly hyperalgetic when handled. There was otherwise no objective disturbance of sensation. The knee- and ankle-jerks were absent

on the right side, while those on the left side were present. The right plantar reflex was not elicited, that on the left side was of the flexor type.

Case 3.—D. B., aged 7 months, a brother of the children whose cases are above referred to, was taken ill on Sunday, September 18th. On that day he was feverish and fretful, and vomited. The fever lasted for three days. His mother said "his head went back too, it affected them all the same." She further stated that "with Willie (D. B.) it was all in the head." On September 20th a convergent strabismus was noted and has persisted since. No weakness of the arms or legs was detected although specially looked for. He seemed to have quite recovered in a few days except for the squint. On October 31st, when examined by the writers together, apart from a slight internal strabismus of the left eye there was no evidence of paralysis. Indeed, he appeared to be perfectly well.

Case 4.—B. A., aged $2\frac{1}{2}$, complained on Tuesday, September 20th, of headache, and was feverish (temperature 101°). On the 21st, 22nd and 23rd, although he seemed "out of sorts" and had no appetite, the temperature was normal and he was not confined to bed. On the 24th, however, he again complained of headache, and the same afternoon of a tired feeling in his legs, while the same day it was noted that the right side of his face was paralysed. During the 25th he was very drowsy, and could only be awakened with difficulty. He was, his mother says, unable to lift his head from the pillow to take nourishment. That night he was very restless and "wandering" (delirious) and he vomited more than once. The temperature on the 26th rose to 104°, but fell to normal in a day or two. The bowels were somewhat constipated throughout. There was no difficulty with micturition. The facial paralysis rapidly improved, and on October 31st had quite disappeared. The patient was then running about quite well.

Case 5.—A. A., aged 5, a sister of the last patient, was taken ill on September 24*th* with headache and a tired feeling in the legs. The temperature was taken and found to be 99.6°. On the 26th and 27th she was running about seemingly quite well. The following day (the 28th) she went for a walk, and on her return her mother says that her legs were trembling and her temperature was found to be 101°. On the 28th, she vomited several times as she did again on the 30th. From her mother's description there would seem to have been some head retraction although she did not complain of

pain in the neck. The right arm was found to be quite limp on September 30th, and her temperature on this day rose to 103°. On October 1st, the legs and right arm were painful. Two days later it was found that both legs were paralysed. Constipation was a prominent feature, and for ten days she had some loss of control over the bladder. The condition on October 31st was as follows :— Both lower limbs were completely paralysed save for slight movement of the toes on the right side, a movement which had only been regained during the past two or three days. There had been pronounced improvement in the right arm, for although all the movements were a little feeble as compared with those of its fellow, abduction at the shoulder was the only movement which was found to be notably defective. She resented passive movement because of the pain which it produced. More especially was this the case in the hamstrings when an attempt was made to extend the legs on the trunk. The muscles were flaccid and there was some wasting. No objective disturbance of sensation was detected. The plantar reflexes and tendon jerks of the lower extremities were absent. The cranial nerves were normal.

Symptomatology.—The character of the symptoms met with in these five cases calls for comment. Two of the cases (A. A. and A. B.) were typical examples of acute anterior poliomyelitis. The remaining three cases were, on the other hand, atypical in so far as B. B. recovered without developing any definite paralytic symptoms, B. A. presented only a temporary facial paralysis, while in the case of D. B. a squint was the only paralytic manifestation. The true nature of the affection might well have escaped recognition had these cases occurred as isolated instances. The circumstance that the five patients were taken ill within a few days of one another, together with the similar symptoms which characterised the onset, makes it certain that all the cases were due to the same casual agent. It may be noted in passing that one of the striking features of recent epidemics has been the comparative frequency of cerebral palsies and of complete recoveries. Thus in the report of the special committee appointed to investigate the New York epidemic in 1907, 27 cases are referred to in which the face was paralysed and 26 in which there was strabismus, while complete disappearance of the paralysis was noted in 5.3% of the collected cases. Again, it should be mentioned that the statistics collected by the New York Committee do not include abortive cases, to which Wickman has especially drawn attention, and under which category the three

exceptional cases above described (B. A., B. B., and D. B.) should probably be placed.

The frequency of pain in the neck, nuchal rigidity and head retraction, of severe headache, and of vomiting account for the by no means uncommon erroneous diagnosis of meningitis in the early days of an attack. The pain and tenderness in the limbs, as exemplified in the case of A. B., may be so severe as to suggest acute rheumatism in the first instance.

An interesting feature in two of our cases (A. A. and B. A.) was the circumstance that the day following the initial rise the temperature fell to the normal, and remained normal for two or three days, the patients in each instance feeling so much better that they were permitted to get up and go about, until the temperature rose again two or three days later.

Contagion and the Incubation Period.—A study of the dates of onset of the disease is most interesting in this connection. The children, it will be seen, were taken ill in the following order, viz. : — B. B. (September 12th), A. B. (September 16th), D. B. (September 18th), B. A. (September 20th), and A. A. (September 24th). Thus it will be noticed that the B.'s were attacked before the A's, and that an interval of from two to four days elapsed between the onset of the disease in each instance. This circumstance certainly gives strong support to the view that the infection was directly communicated from one patient to another. Assuming that the disease is only contagious after the appearance of the first symptom, the data relating to the incubation period are as follows :—

(1) A. B., B. B., and C. B. slept not only in the same room, but in the same bed. A. B. was taken ill on September 16th, *four days* after B. B. It certainly seems very probable that A. B. was infected by B. B.

(2) D. B., who slept in a cradle in the same room as his father and mother, first manifested symptoms on September 18th. Was he possibly infected by B. B. or A. B. ? It is interesting to note that on and after September 14th, *four days* before D. B. was taken ill, B. B. slept in the same room as his mother and D. B. From September 16th onwards A. B., B. B., D. B., and Mrs B. slept in one room, while C. B. and Mr B. slept in another.

(3) Since A. A. and B. A. slept in the same room, and since B. A. was attacked on September 20th and A. A. on September 24th, it seems highly probable that A. A. was infected by B. A. Here again, it is of great interest to note than an interval of *four days*

elapsed between the first manifestation of the disease in these two cases.

(4) Whence came the infection in the case of B. A. ? The A.'s, it will be noted, were taken ill after the B.'s. If, as the evidence certainly seems to forcibly suggest, A. B. was infected by B. B., D. B. either by B. B. or A. B., and A. A. by B. A., it seems only reasonable to conclude, having regard to the time interval, that B. A. was in all likelihood infected by the B. family. Very positive statements were made by both Mrs A. and Mrs B. to the effect that neither of the A. children were in the B.'s house, and none of the B. children in the A.'s house after B. B. was taken ill, *i.e.* after September 12th.

Is there any reason for supposing that the infection may have been carried by a third person ? Mrs A. states that on September 16th, and again on the 18th and 19th, she visited the B.'s house, and that on the two dates last mentioned she remained on each occasion in the house for several hours assisting to nurse the baby (D. B.). This circumstance, when taken in conjunction with the negative facts above referred to, certainly suggests the possibility that she may have carried the infection. If this is so, it is probable that here also the incubation period was four days or less.

Finally, these observations certainly suggest that the incubation period of the disease, in the case of this local epidemic, was probably four days or less.

The scarcity of observations bearing on this question is indicated by the Committee appointed to investigate the New York epidemic of 1907. After reviewing the literature, they conclude by stating that : —" The data on the probable average incubation period are too meagre to permit of definite conclusions, though the evidence indicates an average incubation period of less than ten days." The paucity of data accumulated by the Committee in relation to the incubation period, when it is remembered that their conclusions are based on a total of 752 cases, is certainly striking. We may quote their statement verbatim in this relation. " The following are our own data concerning this point. Case 173 had been in Vermont until ten days before the onset, and Case 123 had also been home from the country for ten days. If the infection resulted in New York, it shows an incubation period of less than ten days. Case 30, living at Dykeman's, had been in New York City one week before. This indicates an incubation period of at least a week, provided infection was contracted there. Case 244 had been home five days

when attacked. If infection resulted at home, it indicates an incubation period of five days or less. On the other hand, Case 214 had been at Old Forge one month when attacked. If this patient became infected before leaving New York, it would indicate an extremely long incubation. In one of the two instances where three cases occurred in one family, the onset of the first was August 25th, of the second, August 29th (night), and of the third, August 30th (noon). It is, of course, impossible to determine whether the subsequent cases were infected from the first case. If this was so, it would indicate an incubation period of four or five days. In the other instance, the first case was a boy, 8 years old, who died on the third day of disease of respiratory paralysis. The next case was a brother, 4 years old, the first symptoms developing on the day the first case died. One week after onset of second case, a sister, aged 6 years, was stricken with the disease. Here again it is impossible to say whether the later cases were infected from the earlier ones. If this should be so, it would indicate an incubation period of three days or less to not over a week."

Wickman's conclusions are referred to in the Report of the New York Committee as follows :—" From a study of the time elapsing between the first and subsequent cases in those instances where two or more cases occurred in the same family or same house, Wickman concludes that the period of incubation of poliomyelitis is usually from one to four days."

Holt and Bartlett have collected reports of 35 epidemics of poliomyelitis prior to the year 1907. In a summary of conclusions they state: " After carefully considering all the evidence brought together in this paper, we cannot resist the conclusion that the disease is communicable, although only to a very slight degree, one of the most striking facts being the development of the second case within ten days after possible exposure."

Etiology.—It is worthy of note that all the children whose cases we have reported were of a robust type, that all were in good health, and that the onset was in no instance associated with coryza or diarrhœa. No abnormal mortality was at the time observed among the poultry or farm stock. Since climatic influences have been regarded as of etiological moment, it may be mentioned that the rainfall in this part of the country was much above the average during the last fortnight of August and first few days of September, while after this date the weather was fine, but not abnormally hot.

The original source of infection is a problem in relation to which

we are not in a position to offer an opinion. Three additional cases of acute anterior poliomyelitis occurred about the same time in the town of Tillicoultry, the dates of onset being September 15th and 20th and October 7th. Two of these cases occurred in the practice of Dr Johnstone, by whose kind permission we were enabled to examine them. The two earlier cases occurred in children, aged seven months and two years respectively. In neither instance was a brother or sister attending school. We can trace absolutely no connection between these three cases and those here reported. Further, we have good grounds for believing that these three cases are the only cases which have been observed in Tillicoultry for some years back. Again, neither the A.'s nor B.'s have, so far as their parents are aware, been in contact with anyone suffering from the disease or, as far as can be ascertained, with any possible carrier of infection There can be no question, however, that cases of acute anterior poliomyelitis have been more numerous than usual in many parts of the country during the past autumn.

Prevalence of acute Anterior Poliomyelitis during the Autumn of 1910

It is generally admitted that acute anterior poliomyelitis is more commonly met with during the late summer and autumn months than at other times of the year, and that in some years these cases are more plentiful than in others. Reliable data bearing on this question are for obvious reasons admittedly difficult to obtain. We have, however, been able to collect some facts and opinions which seem to clearly indicate that, in certain parts of the country at any rate, the disease has been more prevalent during the past autumn than for several years past. In recording the results of our inquiries, we welcome this opportunity of thanking those who have helped us either by placing data at our disposal or by a general expression of opinion. There can be no doubt that cases of poliomyelitis have been more numerous than usual this year in Edinburgh and its neighbourhood. Through the kindness of the physicians to the Edinburgh Hospital for Sick Children (Drs H. M. Dunlop, John Thomson, and J. S. Fowler) we are enabled to cite figures as to the number of cases of acute poliomyelitis treated in their wards during the late summer and autumn months (July to November) for the past five years.

1906	1907	1908	1909	1910
18	10	8	3	23

Our thanks are also due to Dr Dingwall Fordyce, who has kindly placed at our disposal data as to the number of cases seen in the Out-patient Department of the Hospital during the corresponding period.

1906	1907	1908	1909	1910
8	7	7	3	14

We are inclined to think, from conversation with physicians who come in contact with cases of poliomyelitis in consulting practice, that these comparative figures perhaps underrate the relative increase of cases of this disease observed in Edinburgh and its neighbourhood during the past year. Three cases of acute poliomyelitis in adults have been seen by one of us (E. B.) within the last three months.

Dr Leonard Findlay has very kindly furnished us with statistics from the Medical Dispensary Department attached to the Glasgow Royal Hospital for Sick Children, for the months of July, August, September and October, from 1906 to 1910 inclusive.

1906	1907	1908	1909	1910
22	16	14	23	38

It would appear from these figures that in Glasgow also cases of poliomyelitis were last autumn more numerous than usual.

Professor Stalker of Dundee writes :—" Dr Kerr showed a case in a child of 17 months, in which the face was involved, at our clinical club last night. Dr Rogers, who has the children's ward here, said that he had three cases of infantile paralysis in the ward at present, and Dr Fraser, the Infirmary Superintendent, said that there was not a single case all last year. The children's ward has only something like ten beds for medical cases in all, so that about a third of the cases at present are recent cases of infantile paralysis."

On the other hand, Dr Ashley Mackintosh of Aberdeen writes :—" It has certainly not been more common here ; I have never come across anything like an epidemic."

Although no attempt has been made to extend our inquiry to the smaller towns or country districts in Scotland, our impression is that the disease has been at least as prevalent in the small districts as in the cities. Half the cases included under the admissions to the Edinburgh Royal Hospital for Sick Children resided in the surrounding country and neighbouring towns. Dr Reid of Forth and Dr Dickson of Queensferry incidentally informed us that they had recently met with four and three cases respectively, not having seen another case for some years, while Dr Muir of Bellshill, Lanarkshire,

in a letter to Dr Byrom Bramwell, mentions that he saw five cases in the month of June and July 1910.

Dr David Drummond of Newcastle-on-Tyne writes :—" There are no data upon which to base a relative statement. You may, however, take it from me that there has been something approaching an epidemic in the North of England, and that it is not limited to the Newcastle district. I have been struck by the increase in these cases during the last twelve months." In a subsequent letter Dr Drummond refers to a local epidemic in Northumberland. He writes :—" Dr Murray of Haydon Bridge spoke to me to-day (December 2nd) about a small outbreak of anterior poliomyelitis in his neighbourhood. It seems that at one very small village, consisting of six or eight houses, he has had three cases within the last eight weeks."

Dr Bruce Low informs us that " At the Sunderland Infirmary, in a children's ward of sixteen beds, there are at present five recent cases of acute poliomyelitis, and that an unusual number of cases are waiting for admission."

Dr Donald of Carlisle writes :—" We have had a small epidemic of acute poliomyelitis here this autumn."

Dr Jacob has very kindly forwarded us the figures from the Out-patient Department of the Nottingham General Hospital for the months of July, August, September and October ; they are as follows :—

1906	1907	1908	1909	1910
25	21	38	26	48

Dr Jacob informs us that there have been a large number of cases in Melton Mowbray and its immediate neighbourhood recently.

Dr Stanley Barnes of Birmingham writes :—" It is rather curious that you should have written to me just now about poliomyelitis. We have had in this district a moderate-sized epidemic of which you may have seen some note in the newspapers. This epidemic, which occurred chiefly in certain villages in Leicestershire, was diagnosed, as far as the newspapers were concerned, as cerebro-spinal fever. I saw none of the cases in Leicestershire, but I saw three who had had attacks of this disease there, and who were sent to me within the next few weeks. They were all cases of acute poliomyelitis."

Dr Michell Clarke of Bristol, in replying to our inquiries, writes : —" There have been a large number of cases of acute anterior poliomyelitis here."

Dr Dyke Acland writes :—" I have a case under my care in St Thomas's, which is one of eighteen cases occurring recently in a small village in Dorsetshire."

A prominent physician in Manchester writes :—" I have not noted any increase in the number of cases of acute anterior poliomyelitis in my own practice in Manchester (Hospital and private) ; but I have heard that there have been more cases of this disease than usual in Manchester recently." Dr Niven, the Public Officer of Health for Manchester, and Dr Heywood have most kindly provided us with figures which, since they include cases of old standing, some of which may have paid repeated visits to the Hospital, have no direct bearing in the present relation.

Dr W. B. Warrington of Liverpool writes :—" There seems an impression here that infantile paralysis has been more frequent this year, but we have not yet had anything like an epidemic." The Registrar to the Royal Southern, at Dr Charles Macalister's request, looked into the matter and writes :—" No case of acute anterior poliomyelitis has been admitted to the Hospital this year."

The data and opinions which we have received from London do not appear to indicate that acute poliomyelitis has been more frequent in the Metropolis this year than in previous years. Dr R. S. Frew, the Medical Registrar at the Great Ormond Street Hospital, has most kindly made out for us a list of cases admitted during the past eight years. From these figures it would seem that the number of cases admitted this year is very much about the average. Dr Frew points out that there are very obvious fallacies, the admissions depending upon the number of vacant beds, the frequency of other diseases at the time, the inclinations of the physicians who admit cases, and so on. No out-patient figures are available.

Dr G. A. Sutherland, writing of the admissions to the Paddington Green Children's Hospital, says :—" I find that only one case of acute anterior poliomyelitis has been admitted during the stated four months this year. As a matter of fact, we rarely get acute cases into the Hospital."

Dr F. E. Batten writes :—" I quite agree that poliomyelitis has been very prevalent this summer (1910), but I don't think it is more so than the summer of 1904. The summer of 1904 was cold and wet, and the mortality from specific fevers was low and the diarrhœa mortality was very low."

Dr Leonard Guthrie informs us that at the Maida Vale Hospital

for Nervous Diseases during the months of July, August and September, ten out-patients and two in-patients were treated in 1909, six out-patients and two in-patients in 1910. Dr Guthrie writes :—" I have never seen anything supporting an epidemic, but agree that most cases occur in the hot months of the year."

Dr S. A. K. Wilson, the Registrar to the National Hospital, points out that no information with a direct bearing on the point in question can be deduced from the available data.

From these observations, we think we are justified in drawing these conclusions :—

(*a*) That acute poliomyelitis was more prevalent than usual in various parts of England and Scotland during the autumn of 1910.

(*b*) That there are grounds for believing that this increased frequency was as pronounced, if not more so, in the smaller towns and rural districts as in the cities.

(*c*) That in certain parts the disease has been met with in epidemic form.

Epidemic Poliomyelitis and Meningococcal Meningitis. Is there any possible Relationship ?

Acute poliomyelitis and meningococcal cerebro-spinal meningitis resemble each other in that both occur in a sporadic and epidemic form, both tend to affect children, both are often ushered in by similar symptoms, while both appear to be only slightly contagious. They differ notably in that poliomyelitis is a disease of late summer and autumn, cerebro-spinal fever of the winter months. Again acute cerebro-spinal fever and posterior basic meningitis have been proved to be due to the meningococcus intracellularis, while poliomyelitis, though its cause still remains undiscovered, is certainly not due to this organism.

The recent occurrence of these two diseases in epidemic form may be accounted for by coincidence. On the other hand, the occurrence of the unprecedented epidemic of cerebro-spinal meningitis in New York and other parts of America in 1904-5, and the widespread epidemic of poliomyelitis hitherto unknown, in 1907 suggests the possibility of some more intimate association. It is interesting to note that in Edinburgh, Glasgow and other parts of the country cerebro-spinal meningitis was met with in epidemic form in 1906-7, while in 1910 cases of acute poliomyelitis have been more numerous than usual. We know of no evidence, however, which demonstrates

that cases of acute poliomyelitis have been especially numerous in localities where cerebro-spinal meningitis was more frequent, or that the two diseases occurred in epidemic form at the same time and in the same place.

Discussion

Dr Melville Dunlop said he would like, before taking part in the discussion, to express the feeling of pleasure and interest with which he had listened to Drs Currie and Bramwell's paper, and to congratulate them upon the concise and admirable way in which they had presented it to the Society. He fully endorsed the opinion that there had been a sudden and very widespread epidemic of poliomyelitis in this country; his own experience bore this out in a very remarkable way. His Resident Physician (Dr Fraser) had informed him to-day that during the last six months there had been over 40 cases treated in the Sick Children's Hospital, 12 or 13 of them in his (Dr Melville Dunlop's) own ward; whereas during a similar number of months within the last twelve years the average number of cases treated in his ward had been 1 or 2. In private practice also within the last six months he had had 6 cases. As showing the widespread occurrence of the disease, he mentioned that 2 of his cases had occurred in Edinburgh, 1 in Haddington, 1 in Perth, 1 in Peebles, and 1 in the Island of Mull.

The points which had struck him were, firstly, the very prolonged and severe nature of the premonitory symptoms in some of the cases and the impossibility of arriving at an early diagnosis. In one case, the child lay unconscious, speechless and comatose for ten days before the development of the paralysis; in another case, the disease was heralded by violent convulsions and, later, delirium for 4 or 5 days, making one suspect cerebro-spinal fever. In other two cases, one was led to suspect tuberculous meningitis; in one of them one felt almost certain that this disease was imminent—the child complained of headache, had frequent vomiting, intermittent pulse, irregular breathing interspersed with sighs, and frequent flushings; it was only after 8 or 9 days that it was apparent the case was one of infantile paralysis, or, he should rather say, poliomyelitis, because children are subject to a great number of paralytic conditions which might be with equal right described under the somewhat generic name of infantile paralysis.

Then with regard to the distribution of the paralysis in many of these cases. In some of them he had been very much struck with

the widespread nature of the paralysis. In one of his cases the child had been completely paralysed, not only in the limbs but also the muscles of the neck and trunk. In two cases there were bulbar symptoms, in one of them the child had evidently difficulty in speaking, swallowing and breathing. He had been struck with the number of cases in this epidemic, much more than in the sporadic cases, with the paralysis of the trunk muscles. Then, as Dr Bramwell had said, in addition to the paralysis of the limbs, facial paralysis had manifested itself.

Another point, which Dr Bramwell had not touched upon, with which he (Dr Melville Dunlop) had been rather struck, was the age of the patients. The average age in about 70 to 80 per cent. of cases is within the first three years, and in this epidemic a large number of the cases had occurred in children considerably over that period—for instance, out of 5 of his cases, one had occurred in a girl of 11, one in a child of 8, one at 6, one at 5, and one at 4.

The prognosis in this epidemic, in his experience, was very much more satisfactory and encouraging than one generally finds. In the case already mentioned, where the child was completely paralysed not only in the limbs but the back, complete recovery took place in about four months, the child being able to walk about quite freely again. In one of the children with bulbar paralysis, paralysis of the muscles of the trunk and neck, and in whom both arms were paralysed—he thought the paralysis of the arms was perhaps the least common—in the course of six weeks the child was running about perfectly well with the exception of some residual paralysis of the deltoid muscles ; improvement was still continuing and there seemed to be hope that ultimately complete recovery would take place. Altogether the prognosis was more satisfactory and encouraging than one expected in this unfortunate disease.

Dr J. H. Harvey Pirie said he wished to add his thanks to those of Dr Dunlop. The paper had been of special interest to him, because he also had the privilege of seeing with Dr Currie some of the cases in perhaps an earlier stage than Dr Bramwell. He fully confirmed all the remarks, and some of the points were of extreme interest. The symptoms here showed great variability, and illustrated the fact that some cases might occur which would not be recognised unless an epidemic were known to prevail at the time. He could corroborate the opinion that a good many of these cases have occurred in different neighbourhoods ; he had seen three from

Bathgate, and the mother had told him that a great number of cases had occurred in that locality.

If this epidemic was going to recur here in the next hot weather as it had recurred in most other places, as, for instance, in America, where it began one summer, died down in the winter, and became more severe the following summer, it would be necessary to pay attention to early diagnosis. There seemed no doubt, from the experience of the American epidemic, that isolation will be necessary. In Nebraska they had failed to stop the disease except by isolation, and he thought they would have to consider that in this country. The early symptoms, unfortunately, were not such as to make it very easy to recognise the condition; it was only after the development of the paralysis that one recognised it, but that was too late, so that one ought to pay attention and try to diagnose the condition before the paralysis developed.

The symptoms recorded by Dr Bramwell were very suggestive—fever, sometimes vomiting, headache and pain. One used to think of poliomyelitis as being a disease in which there was no pain. But recent experiments have shown that in this epidemic form there is a meningitis to start with which accounts for the pain felt in the back, head and lower limbs.

The rigidity of the back was also a very important early symptom, suggestive of cerebro-spinal meningitis.

The examination of the cerebro-spinal fluid might be of value before the paralysis developed, after that it was no use. On examination of the cerebro-spinal fluid during the incubation period, changes are found; it is under increased pressure, and there are some cells, chiefly mononuclear in type; but these changes disappear after the paralysis develops.

As to treatment at this stage, several observers have noted that urotropine has been of value; it liberates formalin in the cerebro-spinal fluid. Any drug which acts as a destroyer of the virus would be useful.

Dr Dingwall Fordyce said there were one or two points he would like to ask Dr Bramwell about. In the first place he had five cases and the two typical ones occurred in the two eldest children. Is it his experience that in this condition the younger the child the less severely does it affect them? Was it in the sporadic form that the young children had the more severe symptoms?

Then the three young children did not suffer from typical

paralysis of the limbs, but they suffered from cerebral symptoms, was it, in Dr Bramwell's experience, common to get cerebral symptoms unaccompanied by limb paralysis? He noticed that Dr Dunlop, in speaking of Dr Bramwell's paper, said that Dr Bramwell had noted cerebral symptoms with paralysis; he (Dr Fordyce) understood that there were cerebral symptoms without paralysis.

Dr Bramwell, while not being dogmatic on the point, thought that the incubation varied from between four days and less. Flexner, in his experiments in animals, came to the conclusion that the incubation period in animals was extremely variable. Did Dr Bramwell mean them to understand that he would ask them to believe that the incubation period in human beings is more or less a fixed one, with a narrow limit of four or five days?

Dr John Thomson said after the full account of their experience at the Sick Children's Hospital which his colleague, Dr Melville Dunlop, had given, he would not say anything more about the clinical features of the cases observed within the last six months. The only question he would ask Dr Bramwell is in connection with the interesting analogy between epidemics of poliomyelitis and those of epidemic cerebro-spinal meningitis. As he said, there are very interesting analogies, but if he (Dr Thomson) were not mistaken there was one striking difference with regard to the seasonal occurrence. In this country poliomyelitis occurs in late summer and early autumn, and this is not the case with cerebro-spinal meningitis so far as he knew.

Dr W. T. Ritchie said, with regard to the relationship of epidemic cerebro-spinal meningitis and poliomyelitis, he would like to ask Dr Bramwell two questions—(1) Whether the particular part of the country or the closely adjacent neighbourhood was particularly affected by cerebro-spinal meningitis in the recent epidemic? because along the northern coast of Fife there was a great deal of epidemic cerebro-spinal meningitis. (2) Whether he has any information to give from Belfast, because Belfast was the district where epidemic cerebro-spinal meningitis was particularly prevalent; they said they got the infection from Glasgow, Glasgow said they got it from Belfast. Any information he could get regarding Belfast would be interesting in this respect.

Dr Bramwell said there was very little poliomyelitis recorded in London. If he (Dr Ritchie) remembered correctly there was no

epidemic of cerebro-spinal meningitis in London at the time when there was in Glasgow, Belfast and other centres; from his own experience he knew that experimenters in London had difficulty in getting specimens and had to import them from Edinburgh, Belfast, Leith and other places in the north.

The *President* said from his own experience he could corroborate the greater prevalence during the last year of poliomyelitis anterior acuta, and endorse the statements which had been made regarding the clinical symptoms of the epidemic form, which seemed to differ from the ordinary sporadic form. They must, however, conclude that it was the same disease. The communication they had heard to-night seemed to him to be particularly valuable because the cases were so isolated and limited, that enabled one to draw conclusions with regard to the incubation period in a way that, in the great majority of cases, it was impossible to do. The suggestion that the incubation period of these cases seemed to be about four days, was one worthy of being noted at all events. He did not for a moment suppose the writers of the paper intended to say that in other cases the incubation period would be exactly four days. In these particular cases the incubation period was four days or less, and, if so, that is a fact of a very great practical importance, because it is very difficult to arrive at any opinion on that point.

As regards cerebro-spinal meningitis and poliomyelitis he did not suppose the writers of the paper suggested that they were one and the same disease. The organismal nature of cerebro-spinal meningitis had been demonstrated in the cerebro-spinal fluid during life, and there had been no observations to show what the nature of the poison in poliomyelitis anterior acuta is. In New York, where the epidemic was so severe, modern methods had failed to show that any organism could be detected—he thought that was so, but was not sure that he was right. Further information was required as to the nature of the poison in this disease, poliomyelitis anterior acuta. It must be difficult to observe, for many exact observations had been made in New York and elsewhere. A number of organisms had been described by some observers, while others had failed to find any definite toxin which was the cause of the condition.

Dr Edwin Bramwell, in reply, said that he would confine

his remarks to a few of the many interesting questions raised by the different speakers. In this paper Dr Currie and he had made no attempt to consider the subject of epidemic poliomyelitis exhaustively, but had taken up three or four points in relation to which the data they presented appeared to be specially instructive.

With regard to Dr Melville Dunlop's experience, Dr Bramwell had not himself observed, in the cases he had seen recently, that the trunk muscles were more frequently affected than in cases of the sporadic type. As to the occurrence of poliomyelitis in older patients, he had recently seen three cases in adults and two in older children. He believed the prognosis was more favourable in the epidemic type than in the average sporadic case.

With Dr Pirie's remarks as to the advisability of isolating cases when the disease was occurring in epidemics, he entirely agreed.

No treatment was at present known which had a definite influence in arresting the disease during the acute stage.

Pain was a prominent feature in the epidemic type, as it was, Dr Bramwell's experience led him to believe, in adult cases.

In reply to Dr Dingwall-Fordyce, among the cases here reported the two eldest patients presented spinal, the two youngest cerebral, palsies.

Dr Currie and he did not for one moment intend to make the general assertion that the incubation period of poliomyelitis was four days. Their position was that, from a consideration of the, in their opinion, important series of facts which they had brought forward, there were good grounds for supposing that in two or more instances the incubation period was four days or less.

There seemed to be no doubt, as Dr John Thomson had indicated, that poliomyelitis occurred especially in the later summer and autumn, epidemic cerebro-spinal meningitis in the winter months. They did not wish to strain the analogy between the two diseases, at the same time they thought there was reason for believing in the possibility of some subtle relationship.

Dr Bramwell was not in a position to give a definite reply to Dr Ritchie's question.

2. THE MEDICAL EXAMINATION OF EMPLOYEES

By H. G. Langwill, M.D., F.R.C.P., Consulting Physician, Leith Hospital

The practice of making a medical inspection of individuals with a view to the early detection of actual disease or a liability thereto is becoming more widespread within recent years. Medical inspection of school children in this country has now been established by Act of Parliament, and, of course, for many years applicants for the various branches of the Civil Service have had to satisfy a medical examiner before being accepted as servants of the State. The need for a much wider inspection of workmen in all kinds of employment has been borne in upon employers in view of the recent Workmen's Compensation Acts, and more especially of the wide interpretations which have been given in various courts to the term "accident." Now if this need is being felt in the case of workmen employed upon land, it is not to be wondered at that shipowners have found it still more urgently necessary for their employees, there being several reasons why medical inspection is especially necessary in the case of seagoing persons.

1. The essentially hazardous nature of much of the work calls for only men in good condition being engaged.

2. The shipowner is liable for accidents to his employees, not only when at their work, but when they are asleep, theirs being, so to speak, a whole-time employment. Thus, a seaman injured when in his bunk (*e.g.* by a collision) falls to be compensated though he was not "employed" at all, in the ordinary sense, at the time of the accident.

3. Owing to the entire absence, in the great majority of seagoing vessels, of any qualified medical aid, a comparatively slight injury may have serious or even fatal results; even if a correct diagnosis be made the treatment may be wrong, as is so often seen in cases of wounds and fractures. An attempt has been made for some years now to improve this state of matters, so far as Leith is concerned, by giving instruction to officers in ship medicine and surgery, but the contrast between the case of an injured man *at sea*, lying in a dark fo'c'sle, with neither doctor nor nurse of any kind, and the lot of the injured *shore-worker*, removed at once to hospital, with skilled attendance and every appliance and facility provided, is not, I think, sufficiently appreciated.

4. But there is an even stronger reason for medical inspection

of crews, for, under a recent Merchant Shipping Act, the owners are liable for the expense of treatment of *illness* arising in any member of the crew, and if the patient has to be left ashore for treatment they are liable for his expenses till he is landed again in the United Kingdom. The detection, therefore, before the man joins his ship, of any probable causes of disability, is of considerable importance, and all the more when it is remembered that no substitutes can be got at sea for the men laid off by illness or accident. The vessel has to be worked by those who are fit, so that if the others are not to be overworked it is also to the interest of the men themselves (though they cannot always be got to see this) that only healthy men should be allowed to join.

It is with the medical inspection, then, of seafaring employees mainly that this paper will deal, and it may be of interest to relate how the system of inspection was established. In the spring of 1907 the Shipping Federation, which is an association of many of the largest shipowners of Britain, summoned to their London offices a conference of doctors (of whom I was one) from the various large seaports in this country and the Continent. Our duty was to draw up a uniform system of medical examination for all ports, a method of recording the results, and a schedule of the various diseases or surgical affections to be reported upon. In regard to this latter I held strongly that there is not the same need for such a high standard of physical fitness in certain respects (*e.g.* hernia, varicose veins) in the case of men taking short voyages (which constitute a fairly large proportion of the sailings from Leith) as with long trips of weeks' or months' duration, such as the inspecting doctors from ports like Cardiff or Liverpool have more to do with. Thus, in my opinion, a man may be passed for voyages from Leith to Hull, but not from Leith to Honolulu. Difficulties arising from, *e.g.* a truss breaking, have not quite the same risk attached in the case of a man who can be in port in twenty-four hours. Books were prepared by the Federation Office containing on each sheet a list in columns of the various disabilities which might be discovered by the medical inspector, who then puts a cross in the particular column opposite the name of the employee, carbon duplicate sheets being used.

Medical men were appointed by the Shipping Federation at each seaport—there are two at Leith—whose duties are (1) to carry out this medical inspection of crews before they "sign on," and (2) to examine employees who allege they have sustained

injuries at their work. As this latter duty includes reporting upon many *shore* workers, *i.e.* those engaged in handling the ship's cargo, who are *not* medically inspected before engagement, I shall confine my remarks at present to the former, viz., the medical inspection of ships' crews. At the outset I wish to emphasise particularly that it is only an *inspection* or survey that is made of the men, and *not* a medical *examination* of a patient in the ordinary sense. It is thus perfectly different from the medical examination of entrants for the Post Office, Customs, &c. ; no family history or the like is asked—it would not be easily obtained even if asked from a crew of aliens—nor is the urine examined. The main points to which attention is directed are—(1) Eyesight ; (2) Hernia ; (3) Venereal Diseases ; (4) Varicose Veins ; (5) Ulcers or Previous Injuries ; (6) Heart and Vessels ; (7) Giddiness and Fits. I should explain that in the case of trading boats with short trips the crew " sign on " for six months, and are then examined ; for long voyages they " sign on " for each trip. In carrying out the inspection the man is asked to let go his trousers, &c., to his feet, and hold his shirt up to his neck. The heart is auscultated, and aneurysmal pulsation looked for ; then a general inspection is made for hernia, &c., and Romberg's test applied, inquiry also being made as to fits ; then the visual tests are employed.

1. As regards *eyesight testing*, the original instructions for the medical inspectors stated that " only officers and deck hands were to be tested for form and colour tests." Thus firemen and stewards were exempted. So far as colour-vision is concerned, this is only natural, but defective eyesight, at least if marked, in any employee on board ship may have serious financial consequences for the owner, not, of course, from risks to the vessel's safety, but from the liability to permanent blindness resulting from even slight injury or disease in a patient who is quite unable to secure skilled treatment at the proper time. Cases of iritis, corneal ulcers, or even foreign bodies, depending upon the care (!) of a fellow-employee in the stokehole, are more than likely to eventuate in permanent damage to the eyesight, so that, from a compensation-risk point of view, a good standard of eyesight is necessary, even in firemen. Unfortunately, it is, I find, not easy to test firemen by the ordinary types, even when allowance is made for the effects of alcohol and tobacco. It is a common complaint that, owing to the constant exposure to the glare of the furnaces in the dark stokehole, they cannot see letters clearly at a distance. Personally

I am inclined to advise the rejection of a fireman who has practically only one eye available for distance vision, owing to the risk run by his employer of having to compensate him for total incapacity for work should the good eye become markedly defective at sea from accident. There is, moreover, the greater liability of any man with defective vision to ordinary accidents from tripping over ropes, &c., in a bad light. I should here add that the examiner merely can *recommend* rejection of any candidate for any cause; it is open to the captain to take whom he pleases for his crew, but the likelihood of his taking a defective man is not great when the deficiencies are recorded by the doctor and explained to the officer in charge.

As regards officers and deck hands, the Board of Trade at present does not require the eyes to be tested separately (though this will be required after 1st January 1914), and the standard presently required is that more than half the letters in the fifth line of the test Snellen's sheets must be read correctly at 16 feet. Unless I notice that there is apparent inequality of the visual power in the two eyes or any visible corneal injury, I do not test the eyes separately. Sometimes one has a little difficulty with foreigners in regard to reading the types, but a more important point is the case of the seaman—who occurs fairly frequently—who is very defective at making out the test types, and yet is said by his officers, to whom the case is reported, to be "very good at picking up the land or lights at sea." His real *far* vision or "look-out" power is apparently much better than testing by Snellen's types would bring out. I suppose the explanation is that his *light sense* is better than his *form vision*, and that it is the light sense which is relied upon greatly in "look-out" duty. I think this condition is commoner among older men, but not by any means confined to them. Out of 930 men examined in 1910 I find there were 28 noted as "defective," of whom 6 were "rejected" after the officers had the degree of defect demonstrated to them. No one is allowed to be present during the medical examination of the employee, but when defective vision is found its presence is demonstrated to the officer in charge, who is called in for that purpose in order that he may decide whether the man will be accepted by him for service or not.

For testing *colour vision* wools are used, but instead of asking the candidate to select the wools of the same colour as the standard in his hand, my practice is to show him a red or a green

skein,and on his naming this correctly, I then ask him to pick out some more reds or greens (as the case may be), *while I conceal the original skein*. I am quite open to correction on this matter, but my reason for employing the test in this manner is that what is required of the seaman is to pick up a red or a green light *individually* at sea, not *by comparison* with a light of similar colour. I am well aware of the divergence of opinion which exists as to the methods which should be employed in testing the colour vision of persons who have to work with *lights* and not with *wools*, and personally I shall be very glad if anyone present can give me his practical experience of the marine telechrome used by the Admiralty, or the method advocated by Edridge Green ; but what I feel is, that while accuracy in comparing colours may be most important in painters or some shop assistants, what is required of a seaman is that he should be able to recognise at once, *without any comparison*, any single individual light as green, red, or (so called) white. That coloured lights may take different tints in a foggy or misty atmosphere is, I suppose, admitted by all, and the most *practical* method of testing the colour vision would seem to be to try the man at sea with actual lights in various weather conditions, but speaking as a landsman, I trust there is no likelihood of this method of testing being introduced. I make no pretensions to being either an oculist or a physiologist, and shall gladly welcome any suggestion as to how best to test for and eliminate the colour-blind seaman. I notice that Dr Freeland Fergus holds strongly that defective colour sense *per se* is not a source of real danger in navigation unless defective light sense is combined with it.[1]

In 1910 I find 12 men noted as defective in the colour test, 5 being rejected, but, since a large proportion of the crews had been examined more than once, several others had doubtless been weeded out in previous years. It is amusing to find that the stock adjective used by Orkney and Shetland men to describe a coloured wool of whose tint they are uncertain is "mauve," the varieties of mauve being sometimes astonishing. A large proportion of my defective-colour candidates occur amongst men from these islands.

2. *Hernias* occasion the rejection of a goodly proportion of the men. When the hernia is small, and kept up by a well-fitting truss, the man is usually passed (though noted, of course, in the books to prevent his claiming afterwards for its appearance), but I must own I have often some doubts if he should be passed, for

[1] *Glasgow Med. Journ.*, June 1910.

even though the truss be really *worn* (and not, as is too often the case, kept in his trunk) it is not always effective in severe straining efforts, and cannot be replaced at sea if it breaks, and the excessive sweating of a fireman soon rots the covering and rusts the metal. I notice that D. R. J. Collie goes so far as to say (in a paper read at Brussels) [1] that if he were an employer he "would never employ anyone with hernia, even though he wore a truss," and I presume he is speaking of shore labour.

On the other hand, if a radical cure has been performed, should the man then be passed as satisfactory? I am aware that the wearing of a truss after "radical cure" is discouraged by surgeons, but is a man who has had "radical cure" performed really no more likely to develop hernia again than one who has never had a hernia? I must confess I feel inclined to order a man who has had a radical cure performed at least to possess a truss, for use if bulging again begins to show itself (though he may not wear it before this), especially if the voyage is a lengthy one. I shall be glad of advice on the following points, viz.:—(1) Is the risk of strangulation as great in the case of old-standing hernia as in the *newly* developed? (2) Should a man with a small umbilical hernia be rejected? (3) Does strangulation occur at all often in the hernial protrusions at abdominal scars? It is very striking to see the frequency of the signs of the abdominal surgeon at work on the appendix, stomach, &c. In one crew of twenty I noted three appendix operation scars.

3. *Venereal disease* does not bulk so largely as the reputation of seafaring men would lead one to suppose. Out of 930 men examined in 1910 there were only 7 rejections from this cause. Of course these figures refer only to cases of acute disease, and do not include cases of slight gleet.

4. As regards *varicose veins*, it is rather difficult to know how to act. Unless they are very pronounced, the man is generally passed on condition that he wears an elastic stocking, which at least acts as an extra protection from injury if not of any remedial benefit. I should be glad of some surgical opinions as to the question of advising operative treatment in cases where it is more a diffuse enlargement of veins around the calf than an enlargement of the main trunk. The condition is very common, especially among firemen.

[1] "Medico-Legal Aspect of the British Workmen's Compensation Act, 1906." London: Baillière, Tindall & Cox.

5. *Heart and Vessels.*—As regards valvular disease, if compensation is well established I usually pass the man, noting the condition, of course, in the book for references, unless the voyage is a long one, when I advise the man to seek another ship. Aneurysm is really one of the most difficult problems, and all the more since the House of Lords' judgment that rupture of an aneurysm in a man occurring when at work, even though the evidence clearly showed that it might have ruptured when in his bed, entitled the relatives to compensation. I am not versed enough in the law to say whether, in the case of a seaman who sleeps at the scene of his work, rupture occurring when off duty altogether would entail compensation being paid in that case also. The detection of an intra-thoracic aneurysm in its early stages is always difficult and well-nigh impossible in the brief medical survey of a crew, so that it is only the visible pulsation that would call attention to it, unless the coughing induced in testing the man for hernia showed the characteristic sound.

Before passing on to refer to the medical examination of employees who have met with *injuries*, I should like to say a few words on an important allied question, viz., examination of an employee who has been off, through *illness*, to decide on his fitness to resume work. I have not had much experience of this, but the importance of medical reporting on an employee was brought forcibly home to me recently. I was asked to examine on the same day two employees who had been off work suffering from "cardiac debility," according to their respective medical attendants' certificates. My opinion was sought on the question as to whether this condition entailed any special risk to the employer. The reason of their being sent for examination probably arose from the following circumstances:—A man was employed chipping paint inside a vessel's hold, for which purpose he stood on a staging about 4 feet high. Suddenly he was seen to drop his hammer and fall between the staging and the vessel's side, and was picked up dead. The employer's view was that the man fell in the act of dying, but probably because no post-mortem examination was held, and it would have entailed considerable expense to fight the case (irrecoverable if it was gained), I think a sum was paid, though without admitting liability. It is not unnatural, therefore, that an employee who has had "cardiac debility" should be sent for examination before being taken on again, and what did I find? Both were men of about 55, but while the one had had recurring attacks of

dyspnœa and debility for 15 years, with irregularity of cardiac action, the other gave a history of marked anginal attacks of recent date and had a very high tension pulse, his pain coming on with any exertion. I felt bound to advise the employers that as regards the latter man they ran an undoubted risk of his death occurring suddenly at his work or elsewhere, and I shall be glad to hear any opinion of my advice. If " contracting out " was allowed, this is a type of case eminently suited for such treatment, as I know the firm are very averse to throwing the man idle.

As regards examination of employees *after injuries*, I have little time left to speak, but many of those present, especially those acting for insurance companies, must have a much wider experience than mine. Last year I find I reported upon fully 60 cases of injury—several being seen, of course, more than once. I should just like to refer to three difficulties met with, and I place first a very real one, viz., the difficulty of getting the *after-treatment* of the patient carried out when the man has ceased attending hospital. For instance, a man was treated in the Infirmary for a bad compound fracture of the leg. When seen by me nearly six months after the accident he was still using his crutches,[1] never having tried to put his foot to the ground, and he had never been back to the ward since the plaster of Paris was removed. Little wonder the ankle was stiff, and the prospect of resuming work far distant. He was under no medical man's care, for he had no doctor of his own, and I had no power to *treat* him ; his *fracture* having united, the house-surgeon's duties to him, I suppose, had ceased. Now, cases like this occur fairly commonly, and until the law allows part of the compensation benefits to be expended by the employer in getting the man fitted for his work, it seems to me that the only other measure is to try to get the house-surgeon to keep the man still in attendance for the injury until the *after-results* are also removed. He can insist on adhesions being broken down ; it is not likely that the employee will pay for a doctor of his own to come and do this. When the employee is in a Friendly Society this trouble does not arise, but a large proportion of the men I have to report upon are under the supervision of no doctor at all. Another difficulty, more financial than medical, I have met with more particularly in the case of dock labourers injured while handling ships' cargoes. Being largely of the casual labouring class, many of them live in lodging-houses or in very poor quarters, and have

[1] When seen again, eleven months after accident, was still using crutches.

nothing but their half-pay compensation money to live on. The result is that I have not infrequently found a man, who is in poor physical health as well as suffering from some surgical injury, unable to purchase the medicines prescribed for his relief; or, living in a lodging-house, unable to rest the debilitated heart and prevent recurring œdema of the legs. Such cases would really improve rapidly if good feeding and medical remedies were supplied in some institution, and I cannot help thinking that some of them are much more really suitable cases for charitable treatment in hospitals than many of their well-off inmates. The last difficulty I shall refer to concerns seamen especially, and that is the difficulty of starting them after an injury with any kind of "light work." An A.B. must be "able-bodied," or he is not merely of little use at sea, but, from the hazardous nature of much of the work, may be a source of compensation risk to his employers. In spite of partially stiff fingers, for instance, or the loss of portions of fingers, he is quite able for much of the work aboard, but since he cannot, when required, "climb aloft in a howling gale," it means practically that the vessel is shorthanded owing to his presence. Of course a man with such disabilities often becomes a stoker, or can take up work ashore, but if he declines this it is difficult often to certify when he is able to return to *his* employment. For instance, a seaman lost the fourth and fifth digits of the left hand at the first interphalangeal joint through injury with a wire rope; though the rest of the hand is quite good, his gripping power is so lessened that he cannot pull himself up or hang on by a rope, and is thus permanently unable for the whole work of an A.B.

Discussion

Professor F. M. Caird said with regard to hernia it was perhaps wiser for a man with an umbilical hernia to have a radical cure performed. It was not certain, however, that a so-called radical cure was a cure; one eminent surgeon holds that there is no such thing as a radical cure; that, he thought, was over-stating the case; his experience was that in the majority of cases there was a radical cure and a lasting cure. It was difficult to get statistics, especially with regard to hospital patients. One found, every now and again, a patient coming back and saying that he had had another operation done by another operator. Personally he had had a number of men, in private practice, from the army and navy where a radical cure had been performed; these men had carried out their work

and been through campaigns in hilly countries, and he had never heard of any relapses. Although a man had a radical cure it would be advisable to keep a truss in store.

With regard to strangulated hernia, he did not remember having heard of a strangulated ventral hernia. He thought that old hernias with large rings were probably less liable to strangulation than more recent ones with small rings.

The prolonged treatment of patients referred to by Dr Langwill was very difficult in Edinburgh where the surgical beds were always in demand, and where the tendency nowadays was to reduce rather than to increase the number of beds. The patient was sent out before the treatment was completed. In Germany the treatment of these cases was very well done, and the patients set up in a very wonderful way. In Dusseldorf, for example, where the patients are sent in for stiff back, neuralgias, and the like, if they are not improving they are subjected to an interesting test, namely, there is a large square full of sand, in the middle of which there is a hopper without a bottom, and these men are sent to shovel the sand for so many hours a day; it was very remarkable how quickly the symptoms went off, when under other circumstances they would have been wandering about the streets doing nothing and perhaps claiming something for compensation.

Mr Alexander Miles thanked Dr Langwill for bringing this important subject before the Society; it was a subject which was so important and so wide that one was apt to go a little beyond it. He did not think they were discussing compensation at all, but rather the avoidance of it; Dr Langwill's point was that by examining the men before they began to work, they would not have the risk of future claims. He (Mr Miles) thought the time would come when all workmen would have to be examined before beginning work of any kind.

With regard to hernia, decisions had been given showing that the man is entitled to compensation whether the hernia developed before he began work or not; the Courts have decided this again and again, and no insurance company will take a hernia case now. If employers are to keep themselves on safe lines they must reject all men with hernia, with or without a truss. With Professor Caird he had no hesitation in saying that a man should not be accepted until he has had a radical cure performed. Dr Langwill had raised some questions with regard to the various kinds of hernia;

each of these would have to be settled *a propos* of the individual case.

With regard to the examination of cases when the patient is ready to resume work, the difficulties were infinite. One saw day after day men hanging on where before the days of compensation they would have been working within a few days after the accident. The subject was of such importance he was sorry there were not more present.

Dr Macrae Taylor said Dr Langwill had not referred to the teeth. It seemed to him that in workmen perhaps more than other people the question was an important one. They all knew how many diseases were attributable to defective teeth. Personally he had to examine engine-drivers—men of excellent physique—going to India, and often in these men the teeth were defective and he had to refuse to allow them to pass until the teeth were attended to. He should like to know whether this was a question which was considered at all ?

Dr James Ritchie said the Shipping Federation Company for which Dr Langwill acted was entirely different from an ordinary insurance company. In their case there was no competition; they could afford to examine their men and lay down their own rules ; whereas amongst insurance companies there was so much competition they were ready to take many without examination at all. Only when insurance companies are sufficiently hard hit in a certain class of cases will they combine to protect themselves, until then there will be no examination of employees. He had been extremely interested in the paper and in hearing the different opinions, particularly those of Professor Caird and Mr Miles with regard to hernia.

The *President* said he entirely agreed with the view that had been expressed as to the very important nature of this paper. As Mr Miles had said, the subject of compensation was a very very big one, and it was quite impossible this evening to enter into any discussion as to how patients should be dealt with who claim compensation and go on for weeks and months doing nothing when they really should be working ; and, as a matter of fact, as soon as their compensation is settled they return to work. The decision of the House of Lords in the aneurism case could only, from a medical point of view, be termed outrageous—to think a man who has an

aneurism dies while doing a little bit of work and his employer is to be mulcted for killing him. He was glad to hear Dr Chalmers Watson say there was a likelihood of that decision being reversed, but the House of Lords is the final Court, and as to how it is to be reversed unless a similar case is brought forward and carried to the House of Lords, it was of course difficult to see.

As Dr Ritchie had said, insurance companies are different from the Company for which Dr Langwill acted. The insurance company takes over the employer's liability. If he were engaged in business and were an employer and had to be liable himself and not through an insurance company, he would rigidly insist that every one of his workmen had none of these diseases on which decisions had been given—there were several of the same kind. He would not take a man with a valvular lesion of the heart as Dr Langwill seemed to do, as an employer he would be mulcted in damages, because if the man had a strain and the heart gave way the employer would have to pay. But, as had been said, it was an exceedingly big subject, and it would be very interesting at some future meeting of the Society to have the question more thoroughly and effectively discussed; it is one of the subjects which would form a most excellent discussion and debate. Perhaps the secretaries would take a note of that, and next winter they might possibly be able to undertake it.

Dr H. G. Langwill, in replying, said he wished to thank the Society for the extremely kind way in which they had received his paper. It was really a short paper referring more particularly to one or two points and difficulties he had found.

As regards Professor Caird's statement, he was glad to have his opinion that with an old hernia with a large ring there was less risk than with a new hernia with a small ring. He was extremely interested in his remarks about the German Institutions, they seemed to take steps to aid the recovery of the employee. His (Dr Langwill's) chief difficulty was in keeping control over the patient when he had passed from hospital; the hospital, not being a State Institution, has no control over the individual, and nobody else appears to have any power to make the man have his stiff joint moved; he should be glad if his remarks would lead to keeping surgical cases in hospital until they were fit for work and not merely cured.

The subject of heart strain in cases of active sclorosis. with its

sequence, was not considered in his paper. He did not go into these lesser, but doubtless important points, and he had no doubt he had passed these cases often. He had never had to report upon a man who has been off work, or incapacitated for work from heart irregularity.

As bearing on the President's remarks, at the Conference of Medical Men in London, at which Dr Langwill was present, with regard to valvular disease it was at first proposed that these cases should all be rejected ; but it was pointed out that a man on a short voyage who could be treated in the course of a few days might be passed, but that for a long voyage it was inadvisable to pass any man whose heart did not appear to be sound.

As regards Mr Miles' remarks, in talking of hernia he was merely asking for information, he was not sure in his own mind as regards passing or rejecting a man with an umbilical hernia. He had not had a large experience of the condition except in infants and older women, and it did seem rather hard to reject a workman (seaman) for an umbilical hernia which was the result of a strain when an infant.

As regards Dr Macrae Taylor's question about the teeth. The teeth did not matter at all. In the Mercantile Marine they did not take the same interest in the teeth that they did in the Navy. If a man goes to sea with bad or no teeth, it is his own lookout, he has to fend for himself, and if he cannot chew what they give him to eat, it is his loss.

As Dr Ritchie said, there was a difference between the Shipping Federation and an insurance company. The Federation is an association for the defence of shipowners ; they do their own insuring in the sense that nearly all the owners are in the Federation. The Federation does the medical examination and sometimes it fights a case, but in most cases it is really the shipowner who has to carry out his own case. Records have to be kept of all examinations—three carbon copies have to be made, one is sent to the owner, one to the Federation Office in London, and one is kept in Leith. If the owner sees that the examiner advises the rejection of " John Black," he can take him or not as he pleases, but if " John Black " is drowned on the trip, it is the owner, not the Federation, who has to settle the damage.

Meeting VI.—March 1, 1911

Dr Byrom Bramwell, *President*, *in the Chair*

Original Communication

A DISCUSSION ON DUODENAL ULCER: ITS DIAGNOSIS AND TREATMENT

Introduction

By F. M. Caird, F.R.C.S.,
Regius-Professor of Clinical Surgery in the University of Edinburgh

Our knowledge of gastric and allied affections has been of recent years strikingly advanced by the freedom with which abdominal exploration has been practised in order to combat certain symptoms, to substantiate a diagnosis, and to establish a definite line of treatment. Hence our appreciation of the relative value of certain symptoms has been increased, and we are now in a better position than formerly was the case to judge the *rôle* they play in guiding us to a definite conclusion, and we can with greater accuracy recognise special ailments and the treatment appropriate to each.

While the arduous act and the short life might hopelessly delay advance, the co-operation of earnest workers and keen observers is continually requisite to aid the formulation of data on which to base an exact diagnosis.

In requesting the individual experience of our medical brethren on the subject of duodenal ulcer we must bear in mind that our attention should be concentrated upon and mainly limited to the ordinary type of the lesion, with its associated indigestion and complications, as distinguished from those varieties called forth by special forms of trauma and general toxæmia.

Symptoms of Duodenal Ulcer—Diagnosis.—It has been asserted that certain prominent symptoms may be grouped together as peculiar to duodenal as distinguished from gastric ulcer. We must acknowledge our gratitude to the Leeds School for this important symptomatic differentiation, and more especially to Mr Moynihan, who, by his stimulating, dogmatic, and bold pronouncement, has given us a lucid text. Moynihan holds that a careful elucidation of certain subjective symptoms, apart from any objective examination of the patient, may lead us to form a correct diagnosis as to the existence of duodenal ulcer; that is to say, he believes that in typical instances there will be found a long-standing history

of intermittent dyspeptic attacks, aggravated during the colder seasons, occurring chiefly in males from 25 to 45 years of age, characterised by gastric discomfort and pain which follows 2 to 6 hours after the chief meal, is frequently also nocturnal, gives rise to a sensation of hunger, and is relieved by taking food. Hæmorrhage, a late development, takes the form of melæna ; and vomiting, infrequent, becomes more pronounced as stenosis, due to cicatrisation, progresses. Beyond local tenderness present in some cases, we are informed that there is no objective symptom of substantial value, and it is indicated that a test meal usually shows less free HCl than is normal.

We are thus presented with features differing from those commonly occurring in gastric ulcer, where pain arises shortly after taking food, is not alleviated by eating, and is more frequently associated with hæmatemesis and vomiting, and an increased amount of HCl.

While we admire the happy terms and trenchant style in which Moynihan expresses the duodenal syndrome, we are not clear as to the ease with which, from symptoms alone, we can define the position of ulcers which lie in such close neighbourhood to each other as on one or other side of the pylorus. We are ready to welcome and to admit the truth of the general statement, although we encounter cases where, with Moynihan's typical symptoms, there may be no duodenal ulcer, and, again, where there may be ulcer and no symptoms. Do the exceptions prove the rule ?

An illustrative case is as follows :—Mrs. X., æt. 45, developed gastric symptoms in summer 1909. Epigastric pain, of a dull, gnawing character, came on 2½ to 3 hours after meals, and was invariably relieved by taking more food. This pain has persisted for the last eighteen months with varying severity, and latterly she has been able to control it by taking sod.-bicarbonate and Gregory's powder. She has been greatly troubled by nocturnal pain, and has been in the habit of taking some form of nourishment—*e.g.* soup—at 2 A.M., and she frequently eats biscuits in the morning about one hour before breakfast to stave off the epigastric pain. There has been no vomiting. She is fairly well nourished, but has lost much weight.

From a general consideration of this case, and the fact that no epigastric resistance was palpable, and that the test meal contained no free HCl, the correct diagnosis of carcinoma was made. The

disease involved the lesser curvature and pyloric antrum, and was at least one inch remote from the pylorus.

Some of us also can recall cases of perforated duodenal ulcers in which antecedent symptoms were lacking. We may leave to the physicians the important task of indicating the more salient points in diagnosis which enable them to differentiate between the allied forms of ulceration, hyperchlorhydria, and the various types of dyspepsia due to latent or hidden appendicitis and to cholelithiasis, since they are chiefly concerned with the early manifestations of these affections in which there is so much room for the exercise of diagnostic acumen. We believe that with the knowledge we have already gained as to the mimicry which frequently obtains between such troubles, that an application of general principles following on careful examination and consideration of each case as a whole should preclude error even under difficult circumstances. Here we may note that the test meal in duodenal ulcer has generally yielded rather an excess of HCl.

We also look to the physician for information as to the best lines on which to conduct the treatment of duodenal ulcer, since we have no doubt that, in its early stages at least, healing may take place under such measures. The fact that recurrence may occur after an apparent cure by the physician, just as this may also follow after—probably more rarely—successful surgical intervention, arises a suspicion that we have not yet arrived at the primal cause of the malady. It has been recognised that duodenal ulcer is most prone to originate in the first part of the duodenum, and becomes less frequent as we recede from the area which receives the acid outflow from the stomach. We require to know the factors which influence the peculiar sex-incidence, and the cause which leads to the initial death of tissue which leaves an ulcer obdurate to heal as long as it is washed by the acid gastric secretion.

Is the ulcer mainly of embolic, toxic, or traumatic origin? Is the geographical distribution limited by race, country, or dietary habits?

It might be supposed that surgical exploration would always reveal to the experienced touch and sight clear evidence of an existing ulcer. Yet failure in this respect has been followed by actual demonstration of the lesion after death. We have observed much variation in the conditions exposed, chiefly in degree, as would be expected.

I. Silvery white patches, occasionally somewhat stellate in outline, superficial, with the very slightest or no trace of induration.

II. Cicatricial puckerings with adhesions and pouches, along with varying degrees of stenosis. We have only rarely met with crateriform thickening.

III. Large tumour-like inflammatory thickenings adherent to the liver, gall-bladder, and omenta, definitely free from, and not merging directly into, the pylorus. Such conditions may by traction evolve symptoms which complicate diagnosis.

The surgical treatment of duodenal ulcer consists primarily in the formation of an anastomosis between the stomach and jejunum, and usually takes the form of von Hacker's posterior gastro-jejunostomy. The curative effect is ascribed to the local rest given to the affected area. It is practically a system of drainage whereby the irritating gastric contents are diverted from the normal route through the plyorus and duodenum. In addition to this, the alkaline contents of the duodenum carried by the afferent loop mingle with the stomach contents and mitigate their acidity.

It is possible to witness the action of the stomach when disposing of a bismuth meal in the living human subject, and this has been beautifully illustrated by many writers.

We know by experience that the natural functions can be well performed after loss of the pylorus, for example after the operation of pyloro-gastrectomy for carcinoma.

So also it has been shown that the aperture in gastro-jejunostomy may function as a pylorus until such time as the ulcer heals. Moreover, Mauntz cites and figures examples taken from individuals in good health, in whom, after a gastro-jejunostomy, the new aperture preserved its function as the main channel, many months after the ulcer had healed and the pylorus had regained its tone.

We have no desire at present to emphasise the surgical aspects of treatment, nor are we about to detail the various forms of anastomosis which have been advocated, and the reasons for their selection in given cases; nor shall we discuss questions as to the size, site, and suturing of the anastomosis, important though such questions be. As a rule, posterior gastro-jejunostomy is employed as the principal curative agent; but while it is thus regarded, many surgeons are of opinion that with it may be combined some adjuvant attack on the local lesion in the shape of excision of the ulcer, infolding or inversion of the affected area, or even closure of the gastric outlet; others, again, have not hesitated to completely

excise the whole affected duodenal segment, following this with end-to-end suture.

That such measures should become a matter of routine we gravely doubt. Excision in any form means increased risk. In regard to closure of the gastric outlet, we may admit that this might ensure greater rest to the affected area. It is, however, questionable if the gain is so marked as would appear at first sight, since in the presence of a gastro-jejunostomy all the food is apparently transmitted by the new route only, and the pylorus does not permit the gastric contents to traverse the duodenum until healing of the ulcer has taken place. A disadvantage is conceivable that in the event of cure a beneficial return to the normal may be prevented by this antecedent artificial closure of the pylorus.

It may, however, be urged that closure of the pylorus prevents a *circulus vitiosus* developing. The once dreaded *circulus vitiosus* is not met with now in well-planned cases of gastro-jejunostomy, and, besides, there are other factors than a *circulur vitiosus* which makes for bilious vomiting after such operations. It may again be advanced that greater security is afforded to the patient, and that by such additional precautions there is less opportunity afforded for a return of ulceration. We have, however, to bear in mind that under no circumstances, however successful it may be generally, can we regard the treatment of duodenal ulcer by surgical means as absolute. Recurrence may take place—it may be at a different site—and while we happily possess no personal acquaintance with these misfortunes, such instances have been reported. Nor has it yet been established that the many tempting additions to a simple gastro-jejunostomy have lessened this tendency in any marked degree, and we know that return has taken place in spite of their use. We await the verdict of further experience on the value of such operative additions.

The results of operation for duodenal ulcer are brilliant. We cannot recall a single instance where a gastro-jejunostomy for duodenal ulcer was followed by recurrence. A return to the normal standard of gastric secretion has been frequently noted years after operation. On one occasion where we required to reopen an abdomen we observed that a hard fibrous mass which formerly occupied the first part of the duodenum had completely disappeared, and that both the pyloric and anastomotic apertures were patent. We have had no experience of melæna requiring direct ligation of a bleeding point in duodenal ulcer. In one case of the gastro-jejun-

ostomy for a definite duodenal fibrous mass—left undisturbed—the operation was immediately succeeded by severe pain and symptoms which stimulated those of perforation. The pallor, small pulse, and lack of abdominal rigidity gave one a key to the situation, and the evacuation of huge quantities of blood proved what had occurred. The patient did well.

Perforation of a duodenal ulcer exhibits symptoms which, as a rule, differ in degree from those of similar gastric lesions near the pylorus. The history—or it may even be the lack of any antecedent history of indigestion—the right-sided pain and tenderness, may aid in concluding as to the exact diagnosis, although it is not always possible in such urgent circumstances to discriminate between gastric and appendicular perforation, more especially when we bear in mind possible irregular positions of the appendix.

As compared with gastric ulcer, the aperture is usually smaller, there is less escape, and the fluid discharging gives an alkaline in place of an acid reaction. We have a constant guide in the pyloric veins to the relative position of duodenal ulcers.

Closure of the ulcer may of necessity entail a gastro-jejunostomy, and this we think should be carried out forthwith in every case if possible.

Discussion

The *President* said the members would agree with him that they were much indebted to Professor Caird for his admirable paper on the features and treatment of duodenal ulcer, and for his valuable experience so lucidly placed before the Society. They would now be glad to hear any observations on the subject. It was extremely gratifying that on such an unsatisfactory night there should be such an admirable attendance; he was sure the Council would feel that in selecting this subject they had met the wishes of the members, and he hoped there would be no delay in the different speakers getting up to express their views. There were both physicians and surgeons of wide experience present, and he would ask the speakers to keep within the limits of time because many no doubt desired to take part in the discussion. It was a little invidious for the chairman to have to ask one man after another to speak, but perhaps he might ask Dr Russell, a physician, to begin the discussion.

Dr William Russell, in response to the President, said that of recent years there had been a great deal of confused writing on the

subject of duodenal ulcer, and it had almost become surrounded by a mass of confusion and mystery, which Mr Caird's paper and the subsequent speakers might have some effect in removing. He could not do more than attempt to submit some of the propositions which were the result of his own experience. The first proposition was that duodenal ulcers are in all respects analogous to gastric ulcers; there is the acute, chronic, and perforated ulcer. Both duodenal and gastric ulcers are commonly associated with, or preceded by, marked disturbance of gastric function. That, however, like every other proposition in medicine, has exceptions. The commonest form of gastric disturbance is associated hyperchlorhydria. What the precise steps of the ulcerated process may be they did not attempt to say. One point he thought it necessary to bear in mind was that both acute gastric and acute duodenal ulcer may be a very superficial erosion, and that this superficial erosion may be, and often is, associated with hæmorrhage both in the stomach and duodenum. When approaching the question of the diagnosis of duodenal ulcer, one must be able to differentiate between the forms of functional gastric disturbance and be able to satisfactorily treat these various conditions, because on the treatment, and in fact upon the failure to remove symptoms, might hang the diagnosis of the existence of a duodenal ulcer.

An acute duodenal ulcer frequently comes on in the course of an attack of hyperacidity. He repudiated Moynihan's view that hyperchlorhydria has no existence, and that the so-called symptoms of hyperchlorhydria are invariably due to duodenal ulcer. Within the last few months he had had under observation three cases of duodenal ulcer with marked melæna, all of which recovered without any serious difficulty under appropriate diet and medicinal treatment. One patient was a man in active business who could not even go to bed, and in spite of that he made a complete recovery, in the sense that his melæna and other symptoms rapidly disappeared. He held that in acute duodenal ulcer, as in acute gastric ulcer, complete and speedy recovery occurred. The fact that one could arrest the melæna and also the pain coming on two hours or longer after food, was an indication, often a proof, that one is dealing with duodenal ulcer. That brought up the second point, namely, the existence of chronic duodenal ulcer.

Certain persons think that they can readily cure chronic duodenal ulcer as they have readily cured chronic gastric ulcer by gastroenterostomy. One did not wish to be uncharitable, but various evidence

came before one from time to time which seemed clearly to show that many cases of chronic duodenal ulcer are cured which never had any existence; all that has been the matter was really a functional gastric disturbance which had yielded no doubt to appropriate treatment. When he was satisfied that a chronic duodenal or a chronic gastric ulcer existed he did not consider it worth while spending much time in trying to heal it medically; he had spent some weeks over various duodenal ulcers in the hope of success, and probably if the treatment had been carried on for months it might have been attained, but in cases in which one is satisfied that a chronic duodenal ulcer exists, these cases are most promptly healed by having a gastroenterostomy performed.

With regard to perforating ulcers there is really no difference of opinion. A perforation, whenever it occurs, requires laparotomy. Mr Caird and he had comparatively recently had to do with a case of perforating duodenal ulcer in which the earlier symptoms of acute duodenal ulcer had been ignored in spite of melæna; this ulcer had come on after a prolonged period of hyperchlorhydria. His contention was, as he had already said, that hyperchlorhydria is a common precursor of duodenal ulcer, but it does not, of course, follow that in all cases of hyperchlorhydria there is duodenal ulcer.

Dr Dewar (Dunblane), after apologising for speaking at such an early hour when so many eminent physicians and surgeons were present, said he would like to ask Mr Caird if he has noted any history of heredity in his cases? He (Dr Dewar) was intimately acquainted with a family in which the mother died of either a gastric or duodenal ulcer; her family of six grew up to adult life, three suffered very severely from dyspepsia all their lives; out of this family of six there were four children, two of whom had had gastroenterostomy performed.

Dr Russell's remarks he thought of great importance. Personally he had been subject to dyspepsia all his life. The first symptom of ulceration began with melæna, after which he had intervals of very good health, and then had attacks of discomfort, never pain, in which he could hardly digest anything; these attacks were followed by periods of good health in which he could digest anything. He did not believe the ulcer in his case was ever healed from the time it began, notwithstanding the long periods of really good health and comparative freedom from discomfort.

Finally, one night an attack of vomiting occurred and persisted uninterruptedly for nearly a fortnight, until he was operated on.

(The President here asked Dr Dewar if the ulcer was duodenal.)

It was a duodenal ulcer, he believed, but the surgeon was present.

Although he was satisfied that he was really better than he was before the operation, he had not had that entire clear record which Mr Caird described. It was now more than two years since the operation, and in that time there had been two attacks of melæna; he believed, however, that these had been brought on by violent and unusual exercise. He had occasional attacks of spasm and occasionally dyspepsia. He found nothing relieved him better than inhalations of chloroform.

He also asked Mr Caird if he had observed any marked diurnal variation in the amount of urea? Dr Dewar had observed in his own case that it varied considerably from day to day. Also could Mr Caird explain why before the operation he had had to micturate about 4 or 5 o'clock in the morning, and since the operation he had never been disturbed?

Dr G. A. Gibson said from a very large experience of duodenal ulcer he would like to say a few words before he had to go. This morning he had taken the opportunity of going over a large number of cases in his case-books with the assistance of his two house-physicians, and from these notes he found that the general expression, which had been so extremely well given by Dr Russell with regard to hyperacidity, was not borne out by his experience. Most of the cases he referred to were undoubtedly cases of duodenal ulcer; they were seen afterwards; all of them recovered with the exception of one who died from pneumonia. He found hyperchlorhydria varied from .1 per cent to .26 per cent. The amount of free acid varied between .02 (very small) and .18 (extremely high). So that one man's experience, at any rate, shows that the condition of acidity in the stomach varies within wide limits. When there has been recent hæmatemesis or extensive melæna he would never dream of giving a test meal and having the stomach contents examined; the risk was too great.

The possibilities of gastric spasm were very interesting. He would like to ask Mr Caird if, as the result of long-continued spasm of this kind, there was hypertrophy of the pylorus?

With regard to diagnosis, he confessed he still found it very difficult to separate cases of acute and cases of chronic duodenal

ulcer; even the diagnosis of duodenal ulcer, although he had seen hundreds of them, still remained a matter of great difficulty. Often he had diagnosed duodenal ulcer to find nothing of the kind on exploring the abdomen, but had found a chronic appendicitis; similarly he had found the condition present was simply gallstones in the duodenum, and this with the help of all the modern clinical methods. He was, therefore, inclined to doubt greatly the accuracy of the diagnosis. He agreed with Dr Russell that cases were diagnosed as duodenal ulcer where no ulcer existed.

He was very sceptical about the healing of chronic duodenal ulcer; an acute ulcer might heal, possibly sometimes it did heal, but about that he preferred to express no opinion.

As regards treatment, whenever he suspected a duodenal ulcer he called in the surgeon; he believed in surgical treatment at the earliest possible moment. He knew of no measure which was so satisfactory as the complete closure of the pylorus.

(The President here reminded Dr Gibson that he had not given the number of his cases—Dr Gibson agreed that he had not.)

Mr C. W. Cathcart said he had just a few remarks to make about cases sent to his ward during the last thirteen years. These were chiefly cases of perforated duodenal ulcer, and he wished to point out the high mortality. Out of 8 cases, 6 were fatal, 2 recovered. In 5 of the cases there was typical hunger pain 1½-2½ hours after food; in 3 of them chronic dyspepsia; in 1 only slight gastric pain lasting for about a month before the perforation. He contrasted this with perforated gastric ulcer, of which they had had 25 cases in the same period; 12 of these cases died, 13 recovered, making nearly 50 per cent. of recoveries in gastric ulcer as against 25 per cent. in duodenal ulcer.

Then with regard to the symptoms, his experience showed that in the majority of cases there was dyspepsia with pain after food; in one or two cases the pain came on about 1½-2½ hours after food. He wished to emphasise that the fact that the symptoms had disappeared is no indication that ulcers are not present. It was worth while remembering that the fact that an ulcer is not recognised or palpated does not disprove its presence; a small ulcer on the posterior surface might be easily present without being palpable.

Professor Alexis Thomson said he did not pose as an expert on duodenal ulcer; it was only recently that surgeons had come into

contact with that disease. It was very striking on looking up one's case-books to find that one's first notes of these cases was some six years ago. Just as in the case of appendicitis, when it had been about for some years, they had not sized up all that was to be known about it, so with duodenal ulcer. As regards diagnosis there was still great difficulty. Many gall-bladder troubles are found, and the two lesions co-existing lead to great confusion. There is difficulty in differentiating ulcers and lesions of the colon, cholelithiasis and, somewhat rarer, carcinoma; difficulties of all sorts arise in connection with diagnosis.

There were some points, in his opinion, which differentiated duodenal from gastric ulcer. The sex preference is extraordinary—he had only had five female, with something like 50 male cases of duodenal ulcer; these were cases in which the ulcer was seen, not merely diagnosed from the symptoms.

The second remarkable point is the relationship to cancer. One of the common occurrences is to have a chronic gastric ulcer become the seat of a carcinoma; but he questioned if anyone present had seen the transference of a chronic duodenal ulcer into a carcinoma; yet there is the same hyperacidity to be dealt with, which is apparently the most notable feature of both conditions. He had had the unique experience of operating on a duodenal ulcer in a patient with pyloric cancer; the duodenal ulcer was first operated on and the patient's life saved, and then the pyloric cancer was dealt with.

Mr Caird had not, he thought, sufficiently emphasised the diet, which was always an important point for differentiation; if the pain is influenced by eating, the ulcer is gastric, if not it is duodenal. He had seen a patient with duodenal ulcer who could eat lobster salad as well as any of the refinements of diet.

Then a word of two with regard to the perforation of duodenal ulcer. He would like to emphasise the importance of transferring the patient to an infirmary or home immediately; and also that the patient should not be strapped down on a stretcher, but allowed to sit up; if the patient lies on his back the duodenal or gastric contents swing about, whereas if he sits upright they drop right down into the pelvis, which is the right place. He had gone out with a medical man who had a perforated duodenal ulcer, and on going down the steps in front of his house, the patient said he felt the trickling down his pelvis. That statement was thought to be a symptom of neurasthenia, from which he was supposed to be suffering, and almost put them against operation.

As regards closure of the pylorus, Mr Stiles had been doing that a good deal ; but the subject was very new.

Dr J. O. Affleck said this subject was an extremely interesting one and well worthy of discussion by such a Society as this. Even if it is not more than an interchange of opinions and individual experience, it is almost certain to bring forward facts both for practitioners and surgeons and to throw light on obscure matters.

With the main points recorded by previous speakers he was almost entirely agreed ; all that he wished to do was to bring forward a few facts from his own experience, which had not been very considerable, yet not inconsiderable.

With regard to diagnosis, no doubt there are those who seem to regard the diagnosis of duodenal ulcer as by no means difficult. His experience was that there were often very serious difficulties, and he would like to refer to one or two of these. They all, of course, knew that the symptoms of duodenal, as of gastric, ulcer are often of negative value. Sometimes the first indication of the disease is a severe hæmorrhage or perforation. But a more serious difficulty arises from the fact that sometimes the symptoms of duodenal ulcer are masked by those of co-existing disease. He recollected a patient who was admitted to the Royal Infirmary with advanced aortic disease accompanied, as these cases sometimes are, with severe præcordial pain. The patient one afternoon complained of very severe pain, became collapsed, and died a few hours afterwards. The post-mortem examination showed advanced aortic disease and perforated duodenal ulcer. The symptoms of duodenal ulcer were entirely masked in that case. Another instance was that of an infant, nine months old, who showed acute abdominal symptoms ; Dr Affleck was asked by a medical man to see the child just shortly before its death. Death took place, and post-mortem examination revealed in this young infant a perforated duodenal ulcer—a rare, but by no means an unheard of thing. It was impossible in that case to diagnose duodenal ulcer. These were illustrations of the difficulties one must be prepared for, and showed that the diagnosis was not such an easy matter.

With regard to the form of the ulcer, whether acute or chronic, he had seen some striking instances of both conditions. He remembered a case brought into the Royal Infirmary where the patient had vomited up a large quantity of blood and was in an extremely collapsed condition. After a day or two, in which the ordinary

remedies were employed, she had another attack of vomiting of blood and died. Post-mortem examination showed 24 superficial ulcers—20 in the stomach and 4 in the first part of the duodenum.

With regard to treatment, he did not think that all cases of true duodenal ulcer should be at once handed over to the surgeon, particularly cases of recent occurrence. He had seen a great many cases of perfect recovery or, at all events, of good health following where all the symptoms of duodenal ulcer were present. One of these was a case in which the patient was in such a collapsed state that when a surgeon was called in he hesitated to do anything at once in order to see if the patient would rally. The patient rallied and rapidly recovered; he is now in perfect health. Cases treated by the surgeon he had found to be most satisfactory, but still if he had satisfactory evidence that he possessed a duodenal ulcer, he should like to try a little medical treatment before having it operated on.

Mr H. J. Stiles said he felt bound to speak, although, like Professor Alexis Thomson, he did not pose as an authority. Firstly, he would like to say one word about diagnosis. There are certain positions of duodenal ulcer he could not diagnose; he was, of course, only a surgeon, but he might go farther and say he saw a good many cases in which his medical colleagues could not diagnose duodenal ulcer. It was quite easy to see how this should be, because there are some cases which do not give rise to the classical symptoms, and certain other cases which do show the classical form. The commonest of all is the condition in which there is a gallstone which has never given rise to any acute colic, but has set up a good deal of irritation, secondary pyloric spasm, interference with the gastric functions, and ultimately to adhesions in the duodenum. Such a case as this is an example of one midway between the classical type and that in which there are no symptoms. That case turned out to be a pseudo-gallstone, but the gall-bladder was adherent to the duodenum. The patient was cured, but ten months afterwards a gastroenterostomy had to be performed. This morning he had operated on two cases, both supposed to be duodenal ulcer; one was a malignant tumour of the colon, the other was a case of gallstones with adhesions to the stomach and duodenum. A medical friend was responsible for the diagnosis of one; he himself for the other. The patient with the gallstones he had seen a day or two previously; she then had a

great deal of gastric disturbance with a history of hæmatemesis seven years ago, and again seven months ago. This led him to say that he did not look upon hæmatemesis as a pathognomonic symptom of duodenal ulcer. He had operated several times on cases in which there has been distinct hæmatemesis or melæna, but in which there was no ulcer.

With regard to Dr Dewar's remarks. He had had two cases in both of which, in his earlier days, he was asked to operate by the physician because the patients were supposed to have a gastric ulcer. No ulcer was found, and a gastroenterostomy was performed. Again he was asked to operate on cases where there were supposed to be ulcers, and he determined in the next case in which there was a gastroenterostomy that he would not agree to do it unless he was allowed to close the pylorus.

The sooner a chronic duodenal ulcer was operated on, he thought, the better ; the results are immensely satisfactory.

Perhaps he might be allowed to state his position with regard to closure of the plyorus. The majority of authorities laid it down that if you see no possible ulcer, gastroenterostomy is not necessary. He entirely disputed that, and he was perfectly certain that that view would not be held long. He thought they must do something for these poor creatures ; he was safe in saying, he thought, that medicine had not been able to do anything, and these patients became neurotic and a burden to themselves and their friends. If you do a gastroenterostomy in these cases the patient will be no worse off. He had not seen a case in which he had operated for great dilatation of the stomach which had not recovered. In many cases he had had no vomiting at all, the patients have put on flesh, and the stomach symptoms in some cases have completely disappeared. A great deal more might be said on this subject.

With regard to what Dr Russell said about acute duodenal ulcer, he (Mr Stiles) was not a physician and did not know much about it, but he would be rather chary of diagnosing acute duodenal ulcer.

With regard to hæmorrhage, if he had a patient with what Dr Russell calls an acute duodenal ulcer, who had had a severe hæmorrhage, he thought the position the surgeon should take up was that he should not be allowed to have another hæmorrhage.

There is another point, namely, Does gastroenterostomy do any harm ? He remembered the case of a child, who is now 8 years

of age, who, when he was eight weeks old, had a gastroenterostomy done for congenital contraction of the pylorus. In that case he had made inquiries and found the mother has had seven children since, and she assured him that she sees not the slightest difference between the physique, digestion and habits of that child and the other members of the family.

The last point is a suggestion—Why is it that we meet with ulcers in this particular part? It is said that it is the acid which strikes this part. That may be, and probably is, one cause, but is there a predisposing cause, is there a deficiency in the blood supply? The question of the direct blood supply he would like some one to take up and work out.

Then why is the hæmorrhage sometimes so free? There is a very large artery running behind this part of the duodenum; it might be that the blood comes from this artery.

Dr G. Lovell Gulland said he agreed with the various speakers who pointed out the extreme difficulty in diagnosis. The hunger pain was often the only symptom which was present to guide one. He would not go into details about the case which Mr Stiles has referred to; it presented all the features of duodenal ulcer. There was hyperacidity, bleeding, etc., exactly like an ulcer; it did not fit into sarcoma. He had seen two other cases in which there was exactly the same difficulty.

He was very much interested in what Mr Caird said about hyperchlorhydria. He was accustomed to divide his cases into two groups, those which show hyperchlorhydria and those which do not; two-thirds show it, one-third do not. He thought the important point from the point of view of treatment was that better results were likely to be got from medical treatment in cases with hyperchlorhydria, while the cases which did not show it will undoubtedly go to the surgeon. He supposed they were all agreed that some cases are bound to be operated on; cases with any definite stricture, any distension of the stomach, and all cases with continuous hæmorrhage which could not be stopped by medical treatment.

The examination of the fæces is a very important point, because nowadays they possessed reagents of sufficient delicacy to show very small quantities of blood in the fæces. If one had a case showing symptoms of one kind or another and found that it was leaking blood all the time, even although in small quantities, then that case

should be operated on. There are also cases where the patients cannot study their diet—for example, commercial travellers who cannot follow any prescribed diet ; these patients always require operation ; while other people, in the same class of life, who can get their meals regularly need not be operated on. He thought most of the members present would agree with Dr Affleck in desiring medical treatment. All surgeons spoke of gastroenterostomy as the simplest possible procedure ; but serious and often fatal results follow. He could not agree with Mr Caird that recurrences did not take place. (Mr Caird here said he had said they might occur.) He (Dr Gulland) was interested to hear of a case in which Mr Caird had performed gastroenterostomy, in which he declared that there has been no return of the melæna. In that case there had been a return of the melæna, and the patient could only keep himself right by attention to diet. There is a large percentage of cases in which gastroenterostomy is performed in which complete recovery does not occur ; all the same probably in nine cases out of ten the operation is most successful.

Mr Wilkie said he was particularly interested in the etiology of duodenal ulcer, and in what Mr Stiles said at the end of his remarks with regard to the blood supply of the first part of the duodenum. The blood supplying this part of the duodenum is derived from the cœliac axis and is drained back into the same veins as the supply at the pyloric end of the stomach. He had found that minute particles introduced into the right gastro-epiploic vein frequently travel back into the veins in the first part of the duodenum. This observation was made by injections into the living animal.

In connection with this subject no speaker had mentioned, what some authorities have referred to, namely, the connection between appendicitis and duodenal ulcer. Mr Moynihan and Dr William Mayo agree that there is at least some connection between appendicitis and duodenal ulcer ; and now Mr Paterson of London has published a paper in which he states that duodenal ulcer may follow appendicitis. The explanation is that the toxaemia in connection with appendicitis favours duodenal ulcer. Another explanation is that there may be this retrograde venous embolus in cases of duodenal ulcer. He had shown experimentally that an embolus may be very easily diverted into a duodenal or gastric vein, and impacting there may cause a thrombosis and a gastric ulcer. He had seen a case in which a

gastric ulcer apparently followed an attack of appendicitis; the appendix was found healthy, but the omentum was found bound to the abdominal wall over the region of the appendix.

Mr Alexander Miles said he had several times seen patients with severe hæmatemesis, so severe as to bring the patient almost to death's door, who, without any operation, seemed to recover quickly; they remained free from all symptoms for some years, and had no signs either of duodenal or gastric ulcer following. He would like to get the experience of some of the members present who could trace cases of this kind back for many years, and learn if severe hæmorrhages frequently occurred. He had sometimes in such cases delayed operating on account of the patient's low condition, and had hesitated to do so after the patient had recovered to a certain extent.

Dr F. D. Boyd said that most of the speakers had agreed that hyperchlorhydria is closely associated with many of these cases. But as a rule they had some underlying organic condition. What he wanted to recommend to his surgical colleagues was that they should try how far they could influence the hyperchlorhydria by the use of fat. He had had a number of these cases under his care during the last year or two, and had found that one could make the hyperchlorhydria, possibly with duodenal ulcer or gastric ulcer, better if one gave the patient fat before food. One lady he saw the other day, whom he had not seen for more than a year when he put her on olive oil, returned to say that she was exceedingly well as regards her stomach, but that she could not climb hills, she had got so stout. In these cases if you cure the debility, you get rid of the hyperchlorhydria; some cases do persist, but he would recommend to his surgical colleagues olive oil or cream before food. By these means the secretion of hyperchlorhydria is far better inhibited than by giving belladonna, urotropine, etc., in fact, by giving olive oil the pylorus can be relaxed and the hyperchlorhydria got out of the stomach by giving fatty matter when the organ is empty.

Mr J. W. Dowden, as bearing on the etiology of duodenal ulcer, mentioned two cases of hairball which had come under his observation. One was in a child of 5 years of age; the interesting point was that the hair was a loose mass in the larger portion of the

stomach, but as it approached the side of the pylorus the mass became extremely hard. The other case was that of an insane woman who used to eat horsehair, her own hair, blankets or anything she could get hold of. The tumour was diagnosed as various things ; but on examining the abdomen it was found to be lax, and the tumour could be felt. This hairball could be seen in the same situation and presenting the same characters as in the case of the child. The mechanical appearance of these extremely hard masses, in this situation, was extremely interesting.

Dr William Russell asked if he might emphasise the importance of getting a later history in cases of gastroenterostomy. The operation had not been in existence long, and he had seen a number of cases in which there had been a recurrence of the symptoms.

Mr Stiles read a list of questions which had been put to his last 40 patients with regard to the after-history of their cases. From the answers which one patient had given—and most of the replies were similar—it was evident that benefit had resulted from the operation. Nearly all of the cases of gastroenterostomy are satisfactory ; occasionally one hears a patient say that he suffers from a little indigestion, but as a rule there is no complaint of any recurrence.

(The President here said that Mr Stiles had taken 40 cases, and asked how long he had gone back. Mr Stiles said four years.)

Mr A. A. Scott-Skirving said it seemed to him that the most interesting symptom of duodenal ulcer was the absence of any symptoms at all. Of course it is a rare condition to find that, but, as he thought Mr Caird and the other speakers had mentioned, one does get these cases. The point is not so much that one does get symptoms as to try and find out the reason why one does not get them. One could understand how a patient suffering from ulcer in the body of the stomach has no pain, and one found little ulcers in the duodenum without any symptoms at all but when one has a patient with an ulcer and no symptoms at all until the time of the rupture, and on cutting down an ulcer almost as large as a watch is found, then it became a matter of very great interest to try and fathom how this patient developed his ulcer and had it for a long time without any symptoms. The patient he referred to had now got over the operation, and he hoped to show him at the

next meeting of the Society, partly as a case without any symptoms and also for another reason. When the man was well enough to have his history gone thoroughly over, it was elicited that occasionally at work he had had a little feeling of nausea, but he would never go farther than that. He could eat dishes of potatoes without pain, and had never vomited in his life until five or six minutes after he ruptured his ulcer. He would be glad if in his reply Mr Caird would throw out any theory.

Further, why is it that while operating for stricture of the pylorus due to duodenal ulcer there is dilatation of the first and second parts of the duodenum? Was it, he wondered, comparable to the dilatation at the anus in cases of intestinal obstruction a little higher up?

The *President* said they must congratulate Mr Caird on the success of the discussion, which had been a very valuable and practical one. He (the President) had been struck with the very large number of cases of duodenal ulcer which some of the speakers seemed to have met with; it had been talked of as occurring in hundreds of cases; personally he had found it, comparatively speaking, a rare condition. Professor Alexis Thomson had brought forward only some 50 cases, seen in his hospital practice during the last five years, and Mr Stiles had brought forward 40 cases in four years. These two eminent surgeons had only some 100 cases between them. Duodenal ulcer seen post-mortem and seen at operation is very much less frequent and common than perhaps some of them had gathered from this discussion. Of course, as had been well said, many cases are diagnosed as duodenal ulcer where no ulcer exists; that perhaps is the explanation.

With regard to diagnosis, he laid special stress on the sex factor. He always hesitated to diagnose a duodenal ulcer in a woman; it required, he thought, much more definite evidence in a woman than in a man. Cold, he had found, made these cases of duodenal ulcer materially worse. One symptom had not been mentioned, namely, jaundice; he had seen it in a few cases which had been proved by post-mortem examination to be cases of duodenal ulcer.

One question raised by Dr Russell was, in his opinion, of the greatest practical importance, namely, the occurrence of hæmorrhage from probably superficial erosions. In the case of the stomach it was well known that cases of very profuse hæmorrhage are met with in which the surgeon sometimes fails to find any lesion in

the stomach in cases in which there had been a profuse hæmorrhage a few days before the operation. He had seen several cases of this sort, where no lesion was found post-mortem, and where soon after a hæmorrhage the patient has died. It is said, of course, in these cases that the hæmorrhage occurs without any ulceration. At a discussion which took place in Liverpool last June, he asked Dr Hale White if he knew of any means of differentiating hæmorrhage in which there was ulcer and hæmorrhage where there was no ulcer? Dr Hale White said he knew of no means. He thought perhaps the explanation is the one which Dr Russell meant to suggest, and which he (the President) had mentioned to Mr Stiles a few days ago, namely, that one may have a superficial ulcer of the stomach quite sufficient to give rise to a bad hæmorrhage, which heals in a few days, and is not found when the patient comes to be operated on or examined post-mortem.

With regard to operation, his own feeling was that in the first instance—in the first attack—it was as well to give the patient the benefit of medical treatment; but if he himself had a duodenal ulcer, if it did not very rapidly heal under diet and medicinal treatment, he would certainly like to be operated on. He had been so impressed with the number of fatal cases he had seen, either from hæmorrhage or perforation, that he had come to the conclusion that duodenal ulcer, as regards fatality, is infinitely more disastrous than gastric ulcer.

Professor Caird, in replying, thanked the Society for the way in which they had received his paper. It was perfectly evident, taking it all round, that amongst the speakers there is unanimity of feeling in regard to the correctness of Mr Moynihan's remarks on duodenal ulcer. No doubt there were difficulties in diagnosis, and therefore it was a great help to have the picture which Mr Moynihan had figured to go on, however correct or incorrect it might be.

With regard to the question of acidity, the majority of them were of opinion that hyperacidity was by no means a constant or stationary characteristic of duodenal ulcer.

As to medical treatment, there was no reason why it should not be carried out; it was a curative agent.

He was much interested in Dr Gulland's remarks about occult hæmorrhage. He (Mr Caird) had no doubt that occult hæmorrhage would probably help with regard to diagnosis.

With regard to surgical treatment it was interesting to hear the

remarks. So far as he could gather, closure of the pylorus and inversion of the ulcer was not a routine way of dealing with cases.

With regard to the state of the patient after a gastroenterostomy. One very carefully limited one's remarks to duodenal ulcer ; if one had spoken of gastrojejunostomy in general, then, no doubt, many of them could have brought forward cases in which the patient had not been greatly benefited by the operation, but limited to gastroenterostomy in duodenal ulcer he could only agree with Mr Stiles that the results of operation are brilliant. He could not recollect a single instance where a gastroenterostomy, practised for a distinct duodenal ulcer, was followed by return of the symptoms of duodenal ulcer ; all of the cases where there was definitely a duodenal ulcer have benefited by the operation. But there are other cases, similar to those recorded by Mr Stiles ; and gastroenterostomy in cases of neurasthenia is a totally different thing, of which they knew nothing.

Dr Dewar spoke of the hereditary nature of the condition. He could not give any information about that ; but, at all events, he had operated on more than one brother in the same family. Nor could he give any opinion with regard to the urea, or to micturition.

Dr Gibson referred to the spasm. He (Mr Caird) thought there was good reason for believing that there was a reflex spasm. As to the hypertrophy, they were not always certain when the pylorus was hypertrophied.

Mr Cathcart mentioned the relatively high fatality of perforated duodenal ulcer. From the statistics it was very difficult to say that the mortality was higher than in perforated gastric ulcer ; it was a question very largely of the individual cases. Many perforations are subacute, and it is only when general peritonitis is developed that the patient comes under observation, and it is very difficult to express an opinion.

Dr Wilkie had drawn attention to the relations between appendicitis and duodenal ulcer. That is a question which arises occasionally. At present he (Mr Caird) only remembered one instance where a patient suffered from duodenal ulcer who had been operated on about a year or eighteen months previously for appendicitis. It was curious that appendicitis should be so common and gastric and duodenal ulcer so relatively rare if there is a connection between these conditions.

Mr Scot-Skirving's question as to why the duodenum is dilated in some cases of stenosis, he could not answer at all.

As regards the President's remarks with regard to hæmorrhages which occur from mucous surfaces and are not found on post-mortem examination, he could not remember any case of that kind which had proved fatal and showed no gross lesion after death.

Meeting VII.—May 3, 1911

Dr Byrom Bramwell, *President, in the Chair*

I. Exhibition of Artificial Arm

Mr J. W. Dowden showed—

A useful and inexpensive form of artificial arm.

II. Original Communications

1 THE FAUCIAL TONSILS, WITH SPECIAL REFERENCE TO THEIR REMOVAL BY ENUCLEATION

By J. S. Fraser, M.B., F.R.C.S.E.,

Assistant Surgeon, Ear and Throat Department, Royal Infirmary; Lecturer on Diseases of the Nose, Throat and Ear, School of Medicine of the Royal Colleges

The object of this paper is to bring before the Society some considerations in regard to the anatomy, physiology, and pathology of the tonsils, and to give an account of a comparatively recent improvement in our operative procedure for the removal of these structures—an improvement for which we are indebted to the United States of America.

Anatomy

These structures are two superficially placed almond-shaped lymphatic glands, which lie between the faucial pillars. They consist essentially of three elements—(1) a fibrous tissue capsule; (2) lymphoid tissue; (3) mucous membrane (Fig. 5). They may be described as having two surfaces (internal and external), two borders (anterior and posterior), and two poles (upper and lower).

In the submerged variety of tonsils (Figs. 4 and 9), with which this paper mainly deals, the *internal surface* has two parts—I. Below the level of the opening of the supra-tonsillar fossa this surface is covered by squamous epithelium, continuous with that lining the mouth and pharynx. The superficial epithelium passes down and lines the crypts of the tonsil which reach into the substance almost to the capsule. At the orifices of the crypts the epithelium is composed of several layers of cells, but deeper down within the

crypts it consists often of only one layer, and, at the bottom of these involutions, numerous gaps occur in the lining membrane. II. *The upper part* of the inner surface of the tonsil—the velar lobe—is above the level of the opening of the supra-tonsillar fossa, and is imbedded in the lateral part of the soft palate ; it is surrounded by the capsule of the tonsil (Fig. 4).

The external surface is completely covered by the capsule, and lies in close relationship to the superior constrictor muscle. In order to understand the capsule of the tonsil we must refer for a moment to the muscles and fascia of the pharynx. The superior constrictor arises in front from the internal pterygoid process, the pterygo-maxillary ligament and lower jaw, and passes backwards and somewhat upwards to its insertion into the pharyngeal tubercle and the raphé ; internal to this we have the muscles of the Eustachian tube and soft palate above, and below the palato-glossus and the palato-pharyngeus along with the pharyngeal aponeurosis. This latter structure, where it covers the outer surface of the tonsil, forms the so-called " capsule " which sends septa into the substance of the gland interdigitating with the epithelial involutions which form the crypts (Figs. 1 and 2) ; these septa sometimes contain muscle tissue. Between these two—the septa from the capsule and the epithelial invaginations—we have the lymphoid tissue of which the main bulk of the tonsil is composed. In actively functionating tonsils this lymphoid tissue shows germinal centres, but in atrophic tonsils these are not observed.

The *anterior border* of the tonsil lies in contact with the anterior pillar of the fauces, which is composed of the palato-glossus muscle with its covering of mucous membrane, and the *posterior border* is in contact with the posterior pillar containing the palato-pharyngeus. On the outer surface of the capsule, at its lower part, some of the fibres of these muscles interlace, and are joined by fibres from the superior constrictor ; at this point, therefore, the fibres of these three muscles are attached to the lower part of the outer surface of the tonsil capsule (Fig. 2).

Below the *lower pole* of the tonsil lies the base of the tongue, and the lymphoid tissue of the tonsil is prolonged over this region, forming the so-called lingual tonsil. The *upper pole* of the tonsil contains the so-called " supra-tonsillar fossa " ; this fossa is not " supra-tonsillar " at all, but is really intra-tonsillar, because the capsule of the tonsil includes the fossa. Thus if we make a vertical transverse section through the tonsil, including the fossa, we see

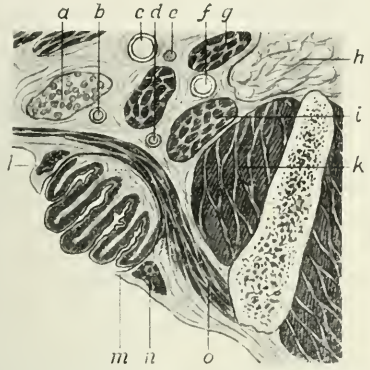

FIG. 1.—Horizontal section through region of the left tonsil (after Cunningham). *a*. Retro-pharyngeal lymph gland. *b*. Ascending pharyngeal artery. *c*. Internal carotid. *d*. Ascending palatine artery. *e*. Glossopharyngeal nerve. *f*. External carotid artery. *g*. Stylohyoid. *h*. Parotid gland. *i*. Styloglossus. *k*. Internal pterygoid. *l*. Palato-pharyngeus. *m*. Plica triangularis. *n*. Palato-glossus. *o*. Superior constrictor.

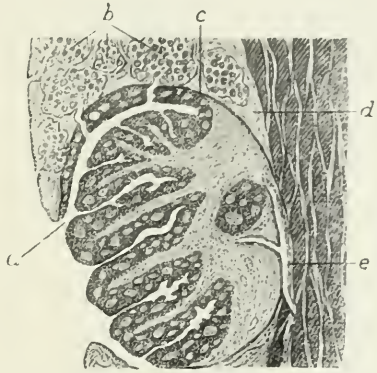

FIG. 2.—Vertical transverse section through region of left tonsil. *a*. Orifice of supra-tonsillar fossa. *b*. Mucous glands in lateral part of soft palate. *c*. Upper part of tonsil capsule. *d*. Peritonsillar space. *e*. Hylum of tonsil.

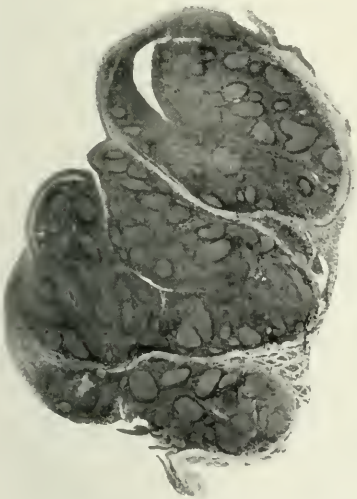

FIG. 3.—Pedunculated left tonsil completely removed with the guillotine. Vertical transverse section × 2 diam. The capsule, which is seen to the right, is entire.

FIG. 4.—Submerged right tonsil enucleated with volsellum and scissors. Vertical transverse section × 2 diam. *a*. Capsule. *b*. Supra-tonsillar fossa. *c*. Upper part of tonsil capsule. *d*. Entrance to supra-tonsillar fossa.

PLATE VII.—TO ILLUSTRATE MR J. S. FRASER'S ARTICLE.

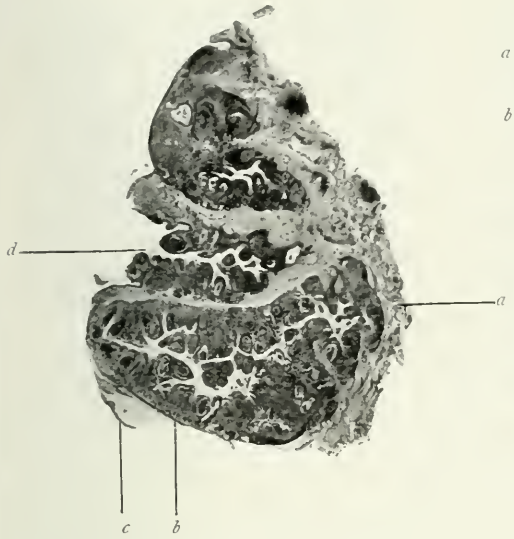

FIG. 5.—Horizontal section of left tonsil removed by enucleation. *a*. Outer part of capsule. *b*. Anterior part of capsule. *c*. Plica triangularis cut through. *d*. Orifice of one of the crypts.

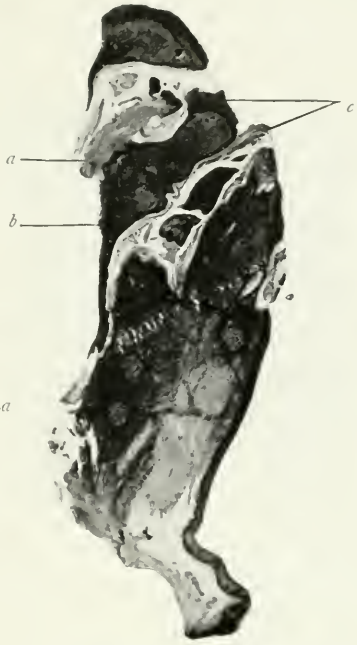

FIG. 6.—Right tonsil from case of keratosis pharyngis. The guillotine operation was performed. *a*. Crypt containing epithelial overgrowth cut through. *b*. Lymphoid tissue of tonsil cut through. *c*. Hyperkeratosis of epithelium lining crypts.

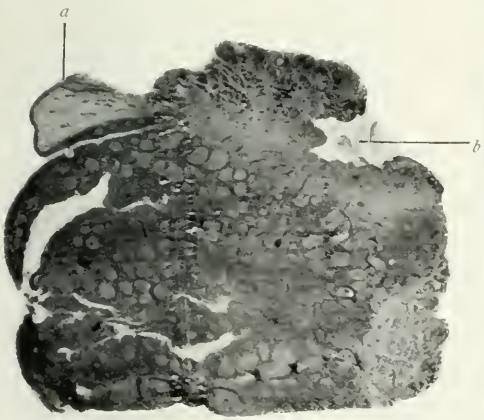

FIG. 7.—Upper part of left tonsil from case of peritonsillar abscess. *a*. Plica supra-tonsillaris. *b*. Peritonsillar abscess cavity.

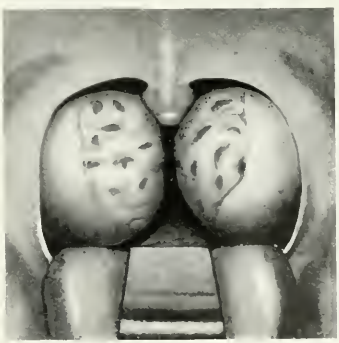

FIG. 8.—Pedunculated tonsils as seen when the patient retches. Type suitable for complete removal with guillotine.

PLATE VIII.—TO ILLUSTRATE MR J. S. FRASER'S ARTICLE.

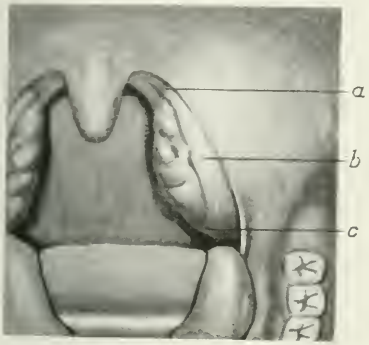

FIG. 9.—Submerged left tonsil. *a*. Plica supra-tonsillaris. *b*. Plica triangularis. *c*. Plica infra-tonsillaris. Note the bulging of the lateral part of the soft palate due to the submerged part of the tonsil.

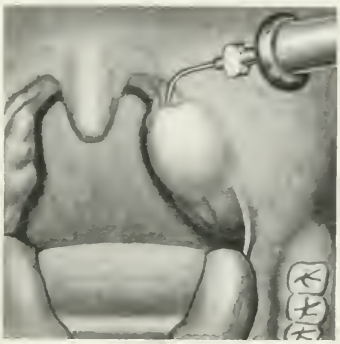

FIG. 10.—Method of injecting the plicæ in cases where local anæsthesia is used.

FIG. 11.—The tonsil is gripped by the volsellum and pulled inwards.

FIG. 12.—Shows the line of scissor cut through the mucous membrane of the plica internal to the anterior pillar. *N.B.*—The cut must not penetrate into the tonsil itself.

PLATE IX.—TO ILLUSTRATE MR J. S. FRASER'S ARTICLE.

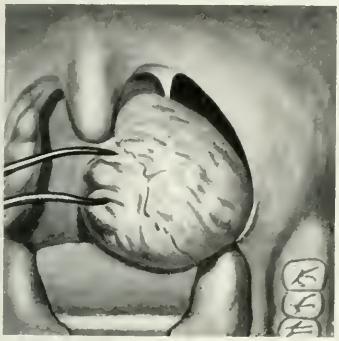

FIG. 13.—The anterior and upper parts of the tonsil have been dissected out. The tonsil is still attached below and behind.

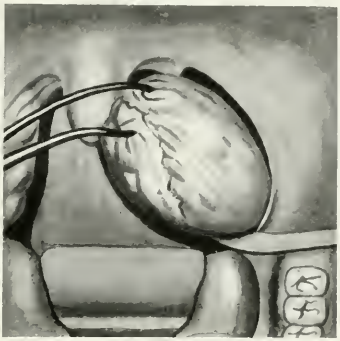

FIG. 14.—The lower pole of the tonsil may be freed in some cases with the curved probe-pointed bistoury. The tonsil itself is pulled upwards, while the knife is passed below it.

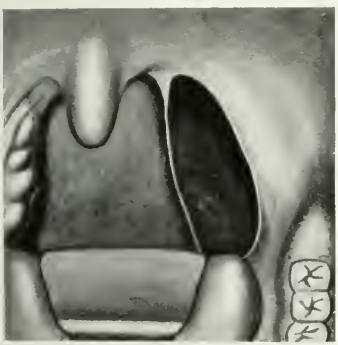

FIG. 15.—The cavity left by enucleation of the tonsil.

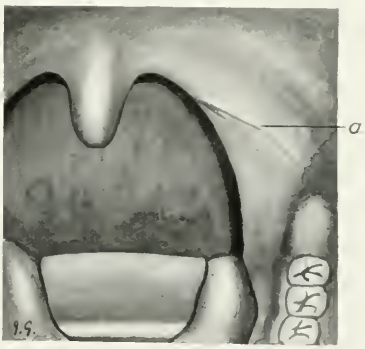

FIG. 16.—The result one year after enucleation. *a*. Anterior pillar flattened out and incorporated with the posterior pillar.

PLATE X.—TO ILLUSTRATE MR J. S. FRASER'S ARTICLE.

that above the tonsil lie the mucous glands in the lateral part of the soft palate (Fig. 2). From above, downwards, we next come to the capsule of the tonsil through which the ducts of some of these glands open into the supra-tonsillar fossa. Between the upper part of the capsule and the cavity of the fossa there is a layer of lymphoid tissue, and, finally, before reaching the fossa itself, we have the layer of epithelium which lines this cavity.

We have already seen that the tonsil is intimately related to two of the muscles of the soft palate, palato-glossus and palato-pharyngeus, the former of which is inserted into the side of the tongue. We must also remember that the soft palate and its movements are intimately connected with the muscles which open the Eustachian tube (the levator palati or levator tubæ, and the tensor palati or dilator tubæ). It is not surprising, therefore, that Reik claims that enlargement of the tonsil may be associated with Eustachian obstruction, not because enlarged tonsils mechanically press on the orifice of the Eustachian tube, but because they interfere with the action of the soft palate, and hence of the muscles which act upon the tube. During the act of swallowing the soft palate is raised, and the superior constrictor and the muscles of the soft palate compress the mucous glands in the lateral part of the velum and force the secretion down the ducts into the supra-tonsillar fossa, and thence into the faucial opening. According to Hett, the upper pole of the tonsil is pressed downwards and inwards during the act of deglutition.

Folds of Mucous Membrane in Connection with the Tonsil.— As before stated, the tonsil lies between the pillars of the fauces, and the mucous membrane which covers these structures is continued into folds as it passes on to the tonsil. Thus above we have the plica supra-tonsillaries, in front the plica triangularis, and below the plica infra-tonsillaris ; these folds are seldom all present in a single case, and even when present they pass one into the other. The best way to understand the relation of the tonsil to the faucial pillars and the mucous membrane folds just described is to compare it to the glans penis surrounded by the prepuce—the pillars, along with the mucous membrane folds, represent the prepuce, and the tonsil, below the level of the supra-tonsillar fossa, corresponds to the glans. In some cases where the upper pole of the tonsil is not strongly developed, and where the orifice formed by the pillars and the folds of the mucous membrane is wide, we notice that when the patient retches the supra-tonsillar fossa is

opened up, or everted, and the tonsil is forced out from between the pillars until it lies on the dorsum of the tongue internal to the isthmus of the fauces (Fig. 8). We are often able to see that the secretion contained in the crypts is extruded by this muscular action. These so-called "pedunculated" tonsils correspond to a glans penis surrounded by a wide prepuce. In other cases, where the upper pole of the tonsil—velar lobe—is strongly developed, and where the orifice above mentioned is narrow, we notice that when the patient retches the tonsil is not protruded between the pillars, but merely forms a bulging on the lateral aspect of the soft palate (Fig. 9). These tonsils have been called "submerged," and correspond to cases of phymosis.

The Peritonsillar Space

This space is situated between the upper part of the tonsil capsule and the inner surface of the superior constrictor, and is occupied by loose areolar tissue. The anterior pillar lies in front of the peritonsillar space, the posterior pillar behind, and above are the mucous glands of the soft palate; below, the space is closed, as before mentioned, by the attachment of the superior constrictor and the two pillar muscles to the outer part of the tonsil capsule. It is in this potenial space (Fig. 2) that suppuration occurs in cases of peritonsillar abscess.

Blood-Supply.—Hett states that the tonsil is supplied mainly by the tonsillar and ascending palatine branches of the facial artery which pierce the capsule at the hilum low down on its outer surface. The tonsil also receives branches from the ascending pharyngeal, the internal maxillary (descending palatine branch), and from the dorsalis linguæ branch of the lingual. The final branches from these vessels pass into the tonsil along the septa sent in by the capsule.

There are two views as to the *lymphatics of the tonsil.* According to one view the afferent lymphatics of the tonsil come from the nasal mucous membrane, so that enlarged tonsils may be regarded as a sign of disease in the nose—v. Lénárt has shown that granular material injected into the nasal mucous membrance passes along lymph channels to the tonsils. According to the other view, the tonsil has no afferent lymphatics unless the gaps in the epithelium lining the crypts can be regarded in this light. The efferent lymphatics pierce the capsule and pass downwards to traverse the superior constrictor, and then open into the superior gland of the

median deep cervical chain, that is to say, the jugulo-digastric or " tonsillar " gland, which, when enlarged, may readily be felt just behind and below the angle of the lower jaw. The two tonsils are connected by lymphatic channels—across the middle line through the soft palate—a fact which explains the frequency and rapidity with which inflammation of one tonsil often follows that of the other.

The tonsillar branches of the glosso-pharyngeus enter below, while the middle and posterior palatine nerves from Meckel's ganglion send branches from above.

Comparative Anatomy and Development of the Tonsil

Ketterer has shown that the tonsils are not vestigial structures. They are absent in reptiles until the crocodile stage is reached, and attain great complexity only in the higher mammals—presumably a recently-acquired organ has some function. Barnes has shown that in the human embryo the tonsils have little lymphoid tissue, and that the epithelium of the crypts is at first similar to that on the surface ; later the central cells of the epithelial involutions break down and are expelled. From the fourth to the sixth months of life lymph follicles begin to show distinctly, and lymphocytes invade the epithelium of the crypts, and, in places, disintegrate its layers. It is these lymphocytes which pass into the cavity of the crypts, and, being frequently retained, from caseous masses. Wood has suggested that the epithelial cells lining the crypts undergo metamorphosis, the result of which is the formation of cells morphologically similar to the lymphoid cells. He also states that transitional cells are found between the epitheliod and lymphoid cells.

Functions of the Tonsils

Rosenheim states that it is generally admitted that lymphocytes are formed in the germinal centres, and that they pass out towards the periphery and force themselves, or are forced, between the epithelial cells lining the crypts. Some writers consider that this process renders the tonsil specially vulnerable, while others hold that it is protective in character. These two views have been termed the " infective " and " protective " theories. Reference has already been made to the opinion of Schoenemann that the tonsils drain the nasal mucous membrane. Spicer regards the tonsil as organs for elimination, while Massini has suggested that they furnish an internal secretion. Fox believes that they are concerned in salivary digestion. A more generally accepted view

is that they are one of the sources of lymphocytes. Gulland is of opinion that they have a bactericidal function. Hendelsohn, Wood, Wright, and others have shown that foreign bodies, such as carmine granules, pass through the epithelium lining the crypts into the substance of the tonsil, but that bacteria are kept back in the crypts. In a recent paper Good states that early immunisation is the function of the tonsils. According to this theory bacteria multiply in the crypts and produce toxines which are absorbed in small quantities ; in this way a certain degree of immunity results. According to Good's theory the mucus in the crypts serves as a culture medium. Dwyer investigated the bacteriology of the tonsil crypts in seventy-two cases, and found one or more varieties of streptococcus in fifty cases, the pneumococcus in fourteen, the micrococcus catarrhalis in twelve ; in sixteen cases diphtheria or diphtheriod organisms were found, in twenty the staphylococcus, in four the diplococcus mucosus capsulatus, and in five the influenza bacillus. Davies examined forty-five tonsils, and in every case found a streptococcus. Good points out that the tonsils probably functionate early in life, and that, relatively speaking, the tonsils are largest up to the fourth year, after which they show a tendency to atrophy. Levinstein has examined hyperplasic tonsils and found active mitosis in the germinal centres, along with an increase in size of these centres, and therefore an increase in the size of the lymph follicles. In hypoplasic tonsils, on the other hand, he found an absence of mitosis, a decrease in the number and size of the follicles, and a tendency of the germinal centres to disappear. Lance does not consider simple hypertrophy of the tonsils to be pathological, but regards it merely as an expression of the power of resistance ; he notes that in young children during dentition the tonsils become larger. Good calls attention to the fact that infants, when attacked by pneumonia, diphtheria, scarlatina, etc., die more frequently than adults, as they have not yet become partially immunised. The conclusion Good comes to is that in children under three years of age the tonsils should not be enucleated.

Diseases of the Tonsils

1. *Simple Chronic Hypertrophy.*—It is no uncommon thing for the tonsils to be so greatly enlarged that they meet in the middle line, and it is easy to understand how in these cases they interfere with respiration, deglutition, and speech (Fig 8). The oropharyngeal cavity is a narrow one, and when the mouth is shut

there is little room between the back of the tongue and the posterior wall of the pharynx.

2. *Recurring Attacks of Tonsillitis, either Follicular or Parenchymatous.*—Rosenheim gives a list of diseases that have been attributed in certain cases to infection through the tonsils. This includes appendicitis, acute rheumatism, arthritis deformans, pleurisy, pericarditis, pneumonia, phthisis pulmonalis, parotitis, nephritis, osteomyelitis, pyæmia and septicæmia. Loeb records four cases of acute hæmorrhage nephritis following tonsillitis. F. Meyer isolated from four cases of rheumatic tonsillitis a diplococcus which produced arthritis in rabbits, while Wagener found the same organism present in the tonsils and in the exudation into the joints. It is noteworthy that the internal remedies recommended in cases of tonsillitis—salicylate or benzoate of soda, salol and aspirin—should be those most generally given in cases of acute rheumatism.

3. *Peritonsillitis and Peritonsillar Abscess* (*Quinsy*).—Patients who are subject to quinsy frequently suffer from attacks every spring and autumn, and sometimes even as often as five or six times a year. Peritonsillitis is, of course, always a result of acute inflammation of the tonsil itself, and Hett is of opinion that peritonsillar abscess is associated with obstruction of the supra-tonsillar fossa and faulty drainage of the crypts which open into it. Infection spreads to the lymphatics of the capsule, and an abscess forms in the peritonsillar space (Fig. 7). On account of the attachment of the superior constrictor and of the two pillar muscles to the lower part of the capsule, the abscess does not burrow downwards, but points, as a rule, through the anterior pillar, though it may open into the supra-tonsillar fossa, or posteriorly close to the posterior pillar. The best method of draining the abscess is through the anterior surface of the soft palate, above and external to the anterior pillar. Frequent attacks of quinsy lead to adhesions between the capsule of the tonsil, anterior pillar, and superior constrictor. These adhesions make enucleation difficult, but the guillotine operation is absolutely useless in such cases.

4. *Tubercular Disease of the Tonsil.*—In regard to the source of infection in cases of tubercular glands in the neck, there is still a considerable amount of dispute, but the tendency is to look upon the tonsil as the original seat of disease. Hurd and Wright found the tonsils definitely tubercular in nine of out twelve cases of tubercular cervical adenitis, and Matthews in five out of eight, whereas in fifty cases in which the cervical glands were not affected

no tubercle was found in the tonsils. Carmichael says that primary tuberculosis of the tonsils is by no means so rare as is usually supposed, and that, out of five hundred tonsils examined by various observers, sixteen (3 per cent.) were tubercular. He maintains that, in cases of tubercular cervical adenitis, the first gland to be involved is the tonsillar gland before mentioned. He himself examined the tonsils from fifty cases; in thirty-seven of these the cervical glands were only slightly enlarged, and in two of the fifty the tonsils were tubercular. Of thirteen other cases with extensive glandular infection the tonsil was found to be tubercular in five, and the proportion would probably have been greater had serial sections been cut. Carmichael thus found tuberculosis of the tonsils in seven out of sixty-three cases, and states that in five of the seven the tubercle was probably primary. He suggests that the source of the infection is probably tubercular milk lodging in the crypts of the tonsil. In a recent paper Leuwer has shown that during the act of swallowing the food passes over the lateral aspects of the pharynx, and not along the middle line; clinical experience in the extraction of fish bones from the faucial region and pyriform sinuses certainly seems to confirm this view. Hett says that in tubercle and sarcoma of the tonsil the capsule is resistant, and the disease therefore tends to spread towards the pharynx, because this is the path of least resistance. According to Carmichael the tubercular tonsil is small and submerged.

5. Such conditions as Vincent's angina, diphtheria, keratosis, tonsil calculus, epithelioma, and sarcoma hardly come within the limits of this paper. Hett states that in the early stages of sarcoma enucleation offers a fair chance of cure.

Indications for Tonsil Operation

In cases of simple chronic hypertrophy, where the tonsils are pedunculated, the guillotine operation is quite sufficient. In cases of recurrent tonsillitis and peritonsillitis, in which the tonsils are submerged, and in cases of tubercular cervical adenitis, enucleation is called for.

Contra-Indications.—It is advisable if possible to avoid operating on the tonsils of singers, especially those whose voices are already trained. Price-Brown states that singing teachers have reported to him that several pupils had had their singing voice practically destroyed by removal of the tonsils. Operation is also contra-indicated in hæmophilia, leukæmia, status lymphaticus, and acute

inflammatory conditions of the tonsils. Dufour states that it is not advisable to operate on the tonsils during the menstrual period.

Treatment of Diseases of the Tonsils

1. *Simple Measures—Gargles and Paints.*—These are of little value, even if the patient is an adept in their use. From what has already been stated in regard to the anatomy of the tonsils, it will be readily understood that gargles and paints can only reach the surface and cannot penetrate deeply into the crypts.

2. *Washing Out the Crypts by Means of a Blunt-pointed Angled Syringe.*—This may be resorted to in the case of singers and of those who, for one reason or another, object to more radical measures. Solutions of argyrol or collargol may be recommended.

3. *Breaking Down the Tonsillar Crypts so as to Allow a more Free Escape of the Retained Secretion.*—Gurich has treated 140 cases of rheumatism by opening the crypts and removing the cheesy secretion. This was followed by an exacerbation of the joint troubles, which this author claims as a proof of the connection between the two conditions. Only twenty-three cases failed to react to the treatment; of the remaining 117 cases ninety-eight were completely cured, and many of these were of long standing.

4. *Cauterising the Crypts.*—This always seems to me a somewhat out-of-date procedure. The crypts go right down almost to the capsule, and it is impossible to be certain that one has entirely closed each crypt from the bottom right up to the mouth. Further, these crypts, which are most difficult to drain, that is to say, those opening into the supra-tonsillar fossa, are inaccessible.

5. *The Guillotine Operation.*—This is the operation suited for those cases of chronic simple hypertrophy in which the enlargement merely acts mechanically (Figs. 3 and 8). For the operation to be successful the tonsil should be of the pedunculated variety (Fig 8). During the year 1910 over 900 cases were operated on in Dr Logan Turner's department by means of the guillotine, whereas only sixty-five enucleations were performed. A modification of the guillotine operation is that in which a volsellum is passed through the ring of the guillotine and the tonsil is firmly grasped and, as far as possible, drawn through the ring of the instrument before the knife is pushed home. St Clair Thomson recommends this method. Whillis and Pybus maintain that they can enucleate 45 per cent. of tonsils with the guillotine. When the ring has been passed over the projecting part of the tonsil they press with the tip

of the left forefinger on the outer part of the anterior pillar so as to force the tonsil through the ring of the instrument. Hett, on the other hand, maintains that only 2 per cent. of tonsils are enucleated with the guillotine. He makes this statement after obtaining a large number of tonsils removed with the guillotine in many of the large London hospitals. I have frequently been informed by a child's parents that " the tonsils have been removed but have grown in again " after the guillotine operation. In cases of recurring tonsillitis and peritonsillar abscess the patient is seldom completely relieved by any operation short of enucleation. Fig. 6 shows the slice that is removed from a submerged tonsil by the guillotine operation. If Fig 6 be compared with Fig. 4 —taken from a case in which the tonsil was enucleated— the greater efficiency of the latter method will be clearly demonstrated.

6. *Morcellement.*—Various cutting forceps have been devised for the removal of the tonsils in small pieces.

7. *Volsellum and Knife.* —This old operation is much more efficient than the guillotine ; the knife should be very sharp. The operation is not, however, based on anatomical study of the tonsils like the following procedure.

8. *Enucleation.*—There are several varieties of this operation. As originally introduced by Pynchon, the operation was performed by cautery dissection, the tonsil being grasped by the volsellum. Others use various forms of knife to dissect out the tonsil. Many American operators prefer the tonsil snare, which is applied after a certain amount of dissection has freed the tonsil from the pillars and exposed the velar lobe. The method I have always used has been that of scissor dissection aided by the finger and, in some cases, by the use of a curved probe-pointed knife liberating the lower pole of the gland and dividing the vessels at the hilum.

Preparation of the Patient.—Williams recommends a hypodermic injection of atropine before enucleation, and Lothrop is in favour of an injection of morphia and atropine. Some operators prepare the field of operation by washing out the tonsil crypts for several days beforehand. This seems to me entirely useless, because if the operation is properly performed the crypts are never reached, as all the cutting should be done outside the tonsil capsule. The only preparation I have advised is the removal of decayed teeth some considerable time before the operation on the tonsils. In this connection it is worthy of remark that Osborne comes to

the conclusion from the examination of nearly 2500 children that enlarged tonsils and adenoids have more to do with cervical adenitis than dental caries.

Anæsthesia.—This operation may be performed either under local or general anæsthesia. The majority of the patients who require enucleation are adults, who, with few exceptions, are able to submit to the operation under local anæsthesia. This method has several advantages: (1) the patient is sitting upright so that there is less hæmorrhage; (2) the patient can also, if necessary, rinse out his mouth from time to time during the operation; and (3) there is no interference with the patient's respiration on account of the tongue falling back and blocking the pharynx. In children, on the other hand, and in nervous adults it is advisable to give a general anæsthetic. In America ether is used almost entirely, but I have not so far employed any other anæsthetic than chloroform, which should be pushed to the extent of abolishing the faucial reflexes. I usually operate by direct daylight, whether the operation is performed under local or general anæsthesia, but reflected light is sometimes a great advantage.

Enucleation under Local Anæsthesia

The patient is placed on a low seat facing the window, and the light falls into his mouth over the shoulder of the operator, who sits on a somewhat higher stool. The faucial tonsils, oro-pharynx, and back of the tongue are first of all sprayed with 10 per cent. cocaine; five to ten minutes later the injection is made. So far I have always used .5 per cent. cocaine to which a few drops of adrenalin have been added (5 mm. to 1 dr.). The injection should be made with a curved needle into the plica triangularis just internal to the anterior pillar (Fig. 10); the needle must only be pushed through the mucous membrane. If the injection is successful the mucous membrane is seen to rise up in the form of a white bleb about the size of a threepenny piece. The needle is now withdrawn and reinserted somewhat higher up towards the junction of the anterior and posterior pillars, and a further injection is made at this point underneath the mucous membrane. In many cases it is also advisable to inject the anterior surface of the posterior pillar. In this way the line of incision is anæsthetised. A similar injection is made on the other side if this tonsil has also to be removed. Yankauer recommends the injection of the posterior palatine nerves on the lines of alcohol injection in cases of neuralgia.

I have tried this method, and the patients when questioned afterwards stated that the pain was much greater on the side on which the attempt had been made to inject the nerves; further practice might, however, render this the more efficient method. As soon as the injection has been completed the operation may be commenced. It is advisable to have a nurse to steady the patient's head and a good assistant to depress the tongue or to exert traction upon the tonsil as may be required. The instruments needed are not numerous—a broad tongue depressor, a uterine volsellum, a pair of blunt-pointed scissors curved on the flat, a long probe-pointed curved bistoury, and some throat sponges in long holders. The left tonsil is firmly grasped by the volsellum, the upper claw of which is inserted into the supra-tonsillar fossa and the other into the lower pole of the tonsil. It is important to take a good grip of the tonsil with the volsellum, for unless this is done the instrument will tear out. One is often surprised, when inward traction is now made on the tonsil, at the large size of the gland as it bulges the lateral part of the soft palate (Fig. 11), and one realises that the part covered by epithelium, and usually spoken of as "the tonsil," really comprises little more than one-third of the whole structure; in fact the first impression one obtains is that one is about to remove the lateral part of the soft palate. The difference between the guillotine operation and enucleation lies in the fact that, in the first case, one can only remove that part of the tonsil which can be forced internal to a line joining the pillars, whereas in the process of enucleation one dissects out the upper pole of the tonsil from its bed and removes the entire structure. One now commences to clip with the blunt-pointed scissors through the plica triangularis just internal to the anterior pillar, beginning below and passing upwards (Fig. 12). *The clips must only pass through the mucous membrane and must not incise the tonsil capsule.* If once the capsule be penetrated it is extremely difficult to get back again to the proper plane of cleavage, and hæmorrhage interferes with the operation. The line of scissor-cut through the mucous membrane is now continued backwards in the arch between the two pillars. All this time strong traction is kept up by means of the volsellum, and after a little scissor dissection the anterior and upper pole of the tonsil begins to appear (Fig. 13). In addition to depressing the tongue, the assistant should retract the anterior pillar with a blunt hook. In many cases one is now able to take a fresh grip of the upper pole of the tonsil and to pull it downwards

and forwards so as to get at the mucous membrane which passes from the tonsil on to the posterior pillar. The scissors are now laid aside for a moment, and the forefinger of the right hand is inserted through the incision into the interval between the tonsil and the anterior pillar and swept round the tonsil from before backwards and from above downwards. If the operator is working in the right plane it is easy to separate the tonsil in its capsule from the superior constrictor muscle unless there have been numerous attacks of peritonsillitis which have caused firm adhesions to arise in this situation, *i.e.* the peritonsillar space. In such cases the dissection must be continued by scissor-cuts alone. This finger dissection is the only part of the operation that the patient objects to, and in many cases it can be dispensed with and its place taken by the scissors. (Lothrop states that the pain after tonsillectomy is most severe in cases in which the finger has been used.) The tonsil is now attached only at its lower part, or, if it has been found impossible to separate the tonsil from the posterior pillar, at its posterior and lower part. These remaining attachments include the hilum at which the vessels enter, and also the lingual prolongation of the tonsil. At this point it may be convenient for the patient to spit out the blood which has accumulated in his mouth and pharynx and to wash out his mouth with boric lotion. Even if the bleeding be severe it is easy to complete the operation with the long curved probe-pointed bistoury. The tongue depressor is again introduced and the tonsil is pulled upwards by means of the volsellum. The knife is inserted below and to the inner side of the tonsil, and, while strong inward traction is kept up with the volsellum, the knife cuts outwards and upwards and divides the lingual prolongation, the vessels at the hilum, and, if it still remain, the mucous membrane attaching the tonsil to the posterior pillar (Fig. 14). If, however, the bleeding be not excessive, the scissors may be used to complete the operation. The tonsil is pulled forwards over the anterior pillar and the mucous membrane attaching it to the posterior pillar is completely divided ; finally the tonsil is pulled upwards while the scissors divide the lingual prolongation and the vessels at the hilum.[1] The patient now washes out the mouth two or three times, and then the right tonsil is dealt with in the same

[1] Of late I have completed the operation with the help of the tonsil snare; this instrument is applied after the tonsil has been freed from the pillars and the upper pole has been well exposed. By this method hæmorrhage is greatly reduced as compared with that observed when the knife or scissors are used to complete the enucleation.

manner. If the operator be ambidextrous the assistant can again depress the tongue while the operator grasps the right tonsil with the volsellum held in his right hand and uses the scissors with the left. If, on the other hand, the operator prefers to use his right hand for the dissection, the assistant takes charge of the volsellum while the operator depresses the tongue with the spatula held in his left hand. As a rule with local anæsthesia there is little or no bleeding at the time of the operation, but the hæmorrhage occurs from half an hour to two hours later, when the effect of the adrenalin has passed off. If the enucleated tonsil be examined at the end of the operation it is not uncommon to find a little muscle tissue attached to the lower part of the outer surface of the capsule at the point where the superior constrictor and the two pillar muscles were attached.

The Operation under General Anæsthesia

After the patient has been anæsthetised the end of the operating table is somewhat lowered so that the head of the patient hangs over, though not to the extent of a right angle with the trunk. The operator stands on the patient's right side facing the head of the table, and the light falls directly into the patient's mouth from the window. It is advisable to have two assistants, one to swab out the blood which accumulates in the pharynx, while the anæsthetist uses the tongue depressor and steadies the patient's head. The gag is inserted and opened, and the left tonsil is dealt with as described above. As a rule, after the removal of the first tonsil the patient is beginning to come out of the anæsthetic; accordingly a small sponge rung out of hydrogen peroxide and held in a long sponge-holder is firmly inserted into the cavity left by the removal of the first tonsil, the mouth gag is closed but not removed, the patient is turned over almost on to his face and more chloroform is given. When the patient is again "under" the head is lowered, the gag opened, the sponge removed, the pharynx swabbed clean, and the right tonsil dealt with as above described.

After-Treatment.—The patient should be kept in bed for at least one day, and should avoid speaking as much as possible; he should rinse out his mouth frequently with boric lotion, phenol sodique, or some other mouth wash. Children who cannot or will not use mouth washes may be allowed to suck lozenges containing formalin. The large cavity left between the pillars (Fig. 15)

usually fills with blood clot within a few hours of the operation, and this clot may still be seen on the second day, towards the end of which it usually comes away, and the raw surface is thereafter covered by a greyish-white membrane, which must be mistaken for diphtheria. There is, as a rule, considerable pain on swallowing for four or five days after tonsillectomy, and, as is usually the case in dysphagia, the patient finds that thickened soups and milk puddings are most easily dealt with. Pynchon advises that the patient should sleep with his head well elevated for two or three nights after the operation, that he should use a gargle every quarter of an hour, and that the wound cavity should be sprayed out every hour with hydrogen peroxide. Although in my experience of one hundred cases I have had two of moderately severe hæmorrhage, I have, so far, had no case of sepsis following tonsillectomy, and accordingly I do not think these troublesome measures are necessary. An inhalation of benzoin from a jug of hot water may, however, relieve the sore throat. The final healing after enucleation occurs in some cases by the growing together of the two pillars from above downwards, thus narrowing the arch of the fauces. In other cases the anterior pillar becomes flattened out and passes into the posterior pillar (Fig. 16). Patients upon whom the operation has been performed say that they find no inconvenience in talking or swallowing, but, so far, I have not performed the operation upon a singer, so that I cannot state the effect on the voice. In order to obviate the growing together of the pillars, Pynchon recommends that the space between them should be massaged with cotton-wool swabs soaked in perchloride of iron and glycerine.

Difficulties and Dangers

1. *Friable and Adherent Tonsils.*—As before stated, the two important things are to grasp the tonsil firmly and to keep outside the tonsil capsule. If the tonsil be very friable the volsellum is apt to tear out however firm a grip be taken, while in patients who have suffered from peritonsillitis the tonsil is firmly fixed and cannot be drawn inwards to any extent with the volsellum. In these cases one is specially apt to clip through the tonsil capsule, and so render complete enucleation almost impossible.

2. *Trismus.*—A short time ago I operated upon a patient who apparently could not open his mouth wider than half an inch; it is, of course, a well-known fact that peritonsillitis is often associated with a certain amount of trismus.

3. *Respiration.*—Under general anæsthesia there is sometimes difficulty with the respiration, but this can be overcome by pulling the tongue and lower jaw forward.

4. *Status Lymphaticus.*—The danger connected with the condition known as status lymphaticus must not be forgotten. Harris and Packard record deaths from this cause.

5. In cases under local anæsthesia the patient may suffer from slight symptoms of cocaine poisoning (breathlessness, palpitation, and faintness).

6. *Hæmorrhage.*—Bleeding may occur at the time of the operation, especially in cases where general anæsthesia is employed, or may come on within a few hours (reactionary) or later (secondary hæmorrhage). Frequent vomiting of blood some hours after the operation shows that hæmorrhage has been going on. The most difficult points at which to arrest bleeding are (1) low down in the cavity near the base of the tongue, and (2) high up in the roof. Ard has collected eighty-five cases of alarming hæmorrhage after removal of the tonsils and adenoids by various methods. Death occurred in fourteen ; in one case only did the hæmorrhage come from the internal carotid artery, but in this case the vessel took an abnormal course. The tonsillar branch of the facial artery was the source of bleeding in almost all cases. These eighty-five cases are, of course, only a small proportion of the fatal cases that have really occurred. It is advisable in every case to inquire as to hæmophilia, and if there be reason to suspect the presence of this disease, it is advisable to avoid operation, or at least to give lactate of calcium for some days beforehand. In regard to the treatment of severe hæmorrhage, the first thing is to apply pressure to the bleeding surfaces. The patient should sit up facing the light, and may be advised to clasp his hands on the top of the head. A small sponge or gauze swab wrung out of peroxide of hydrogen and held in a long sponge-holder should be pressed into the bleeding cavity. Pressure should be kept up for five or ten minutes. If the bleeding recurs we may try the effect of turpentine applied in the same way. Adrenalin is of no use in these cases. The patient should sit up as long as possible, and an injection of morphia is an invaluable adjunct to treatment. Should this fail, an attempt may be made to catch the bleeding vessel. Lothrop advises that numerous small swabs on fine holders should be used to fill the cavity left after the removal of the tonsil, and that these should be removed one at a time, so that the bleeding vessel may be seen and picked up.

It is important also to retract the anterior pillar with a blunt hook in order to see the bleeding point. Special forceps have been devised by Rosenheim for ligaturing a bleeding vessel in the bed of the tonsil, but this procedure is by no means easy. If we fail to ligature the vessel, the forceps may, however, be left in position. The third method is the suture of the palatal arches. This may be done either with a curved needle in a needle-holder, or by means of steel clips and special forceps. I have not yet had occasion to use this method, but if I do I shall attach silk thread to the middle of each clip, in order to prevent the clips from passing into the food or air passages. Various forms of tonsil compressor and clamps have been devised ; one blade fits outside over the angle of the jaw, the other fits into the cavity left by the removal of the tonsil. Ligature of the external or common carotid artery is the last resource.

7. *Sepsis.*—This may manifest itself in various forms—(*a*) diphtheria ; (*b*) septic infection of the cervical glands ; (*c*) purulent otitis media and mastoiditis (Pairy) ; (*d*) Parish records a case of surgical emphysema following removal of tonsils, and Stewart one of extravasation of blood into the tissues of the neck, cheek, and chest twenty-four hours after the removal of the tonsils. Pierce gives an account of a case of torticollis following enucleation ; (*e*) nephritis ; (*f*) septic rashes, acute articular rheumatism (Ard), chorea and endocarditis have been recorded as following tonsil operations ; (*g*) septic thrombosis of the internal jugular vein ; (*h*) septicæmia. Wishart records a case apparently of this nature. Dean records a case of death from meningitis following tonsillitis.

It must not be supposed, however, that hæmorrhage, cervical abscess, submucous septic œdema, etc., do not follow tonsillar infection apart from operation. Newcombe reports a case of hæmorrhage following quinsy. The common carotid artery was ligatured and the patient recovered. The same writer has collected forty-one cases of hæmorrhage in connection with tonsillar suppurations. In twenty-six of these the abscess opened spontaneously, and the hæmorrhage was fatal in fifteen ; in fifteen cases the abscess was opened by incision, and eight of these patients died. As before stated, in my own cases of tonsillectomy, numbering a hundred and twenty (thirty under chloroform, ninety under local anæsthesia), I have not had any cases of sepsis, but have had two of moderately severe hæmorrhage. The local application of a sponge wrung out of turpentine was sufficient to control the bleeding on both occasions.

In conclusion, I wish to tender my thanks to Dr Logan Turner

for the great majority of the opportunities I have had of removing the tonsils by enucleation; also to the authorities of the Royal College of Physicians Laboratory forthe means of doing microscopical work; and, finally, to the Carnegie Trustees for their kindness in providing the illustrations in the text.

Discussion

Dr Logan Turner said there was an old story told of the late Professor Ashton Flint, one of the greatest leading physicians some years ago. He was examining a candidate at the Bellevue Medical College, New York, on physiology; he said, "What is the function of the spleen?" The candidate answered, "To enlarge in malaria." "And what is the function of the faucial tonsil?" "To inflame and suppurate in quinsy." And Flint said, "You have done a very good examination, for your knowledge on this subject is as good as mine." Now, however, they knew more than they did at that time.

The question, When is a tonsil not a tonsil, was not such an easy one. One could not tell on looking at the tonsil whether it was doing its duty or not. The larger the tonsil the safer, the small submerged tonsil is the dangerous one.

If he might throw out a suggestion to the secretaries of the Society, if they ever were in want of a subject for discussion, he would say that an excellent one would be "The upper air passages as portals of entrance for general infection."

The operation under discussion he liked to divide into two, namely, the operation in the child and the operation in the adult. In the great majority of cases in children he would throw out a plea for the guillotine. In a child the question of tuberculous infection through the tonsil had to be considered. A great deal of work had been done on this subject. Fraenkel's statistics showed 5 per cent. of primary infection of the tonsil; Jude says that after examining several hundreds of tonsils he only found tubercle in 1 per cent. In other words, then, primary infection of the tonsil is not so frequent as might be supposed.

Now from the clinical side it was found that in 95 per cent. of tonsils there was some enlargement of the glands, but how often are these glands tuberculous—rather, how seldom are they tubercular? Over and over again enlargement of the glands disappears after the guillotine has been applied. Therefore in the child, except in

exceptional cases, he did not think the use of the knife was required. The guillotine operation is simple, a little gas or ether is given, and the child is up and about the next day; years afterwards a large number of them are found to be perfectly well.

In the case of the adult the conditions are quite different. The tonsils are usually removed in an adult because of recurrences of quinsy or on account of toxic symptoms.

Dr Fraser's work, like all his work he brings before this Society, is of a high order, and as he has shown that the supratonsillar fossa is higher than the soft palate, therefore the only operation is enucleation. In some of these cases the crypts run out to the capsule or almost to the capsule, and there is no use slicing a tonsil, if you want to the crypts you must enucleate. Therefore in children, except in tuberculous cases, the guillotine; in adults, enucleation.

As regards technique, personally he had given up using cocaine; he injected novocaine and a few drops of adrenalin.

With regard to hæmorrhage, if the whole tonsil is thoroughly removed there is no hæmorrhage; it is because a piece of the tonsil is left that hæmorrhage occurs; it is the stump that bleeds.

Patients do not get over enucleation so readily as they do the simple operation. He had found the application of hot fomentations relieve the patient very much.

Dr Elliott, U.S.A., said he would like to say a word or two, as he came from the country where the operation of enucleation originated, and is adopted by most men in America. The method described by the reader of the paper is the same as that used in America, but there they nearly all used the snare, which was more advantageous than the scissors. If Dr Fraser would just try the snare, he was sure he would give up the scissors, as the snare was a great improvement. With regard to enucleation he thought it far superior; he was very much in favour of it and used it almost entirely with splendid results.

Dr Porter said with regard to technique, it is simple if one manages to strike the right depth, if one does not get outside it is rather a difficult operation to do. The great point is to get hold of the tonsil, getting hold of the anterior pillar, and if that is done one almost invariably hits on the right depth. If this is done, he did not see why the snare should be superior to a pair of scissors.

As regards the indications, he agreed with Dr Turner that in the vast majority of children, if it is necessary to touch the tonsil, the guillotine is all that is necessary.

Dr J. D. Lithgow said that in some cases where the tonsil does not project a punch could be introduced and was better than scissors.

Mr Struthers said that some twelve years ago he had had the privilege of working in private practice with Dr Maclaren of Carlisle and was well acquainted with his operation. The results obtained, as far as could be seen, were good, but at the same time the operation struck one as being a severe one; the patient required to remain in bed several days and lost a great deal of blood.

He would like to ask Dr Fraser and Dr Turner whether they had any trouble with their local anæsthetic. It had struck him that the solutions used are too strong. Half per cent. novocaine was given in ordinary work, why not in the case of the tonsil? He thought it would be sufficient to give a 1 in 500 solution. He thought he was right in saying that patients were apt to get a little faint, and it has seemed to him that that might be avoided by the systematic use of a very much weaker solution.

Mr Dowden said although he was a surgeon who had given up operating on the tonsils, he would like to say a word or two. Dr Fraser had mentioned the friability of the tonsils. On account of the friability the late Professor Annandale adopted a method of lacerating the capsule of the tonsil—the capsule was torn with the fingers and pushed forwards, and with a large sharp spoon rapidly removed. He (Mr Dowden) was not acquainted with the scissors at all.

He would like to ask Dr Fraser with regard to hæmorrhage. When hæmorrhage is sufficiently bad to necessitate calling in a surgeon what are the best means to be adopted? Dr Fraser mentioned pressure, but when one is called to a patient, often in a dark room, and one has to get inside the mouth, one is apt to destroy a good deal of tissue about the tongue. If it is necessary to ligature the carotid, is it the common carotid or external carotid which is to be ligatured? He mentioned the case of an old man with a traumatic injury at the angle of the jaw with severe

bleeding in which he had ligatured the common carotid, and in which the patient got quite well.

Dr J. S. Fraser, in reply, said he was much obliged for the way in which the Society had received his paper, and for the discussion on it.

With regard to Dr Turner's remarks that the child should be guillotined and the adult's tonsil enucleated, he thought both Dr Turner and Dr Porter had misunderstood his attitude with regard to the operation in the child. He did not say that all tonsils should be enucleated, far from it; what he said was that if the tonsil calls for operation, the removal should be done thoroughly. If the tonsil is healthy and causing no symptoms, leave it alone; what he did not approve of was taking a little bit off the top. If a tonsil is peduncular the guillotine is indicated; if submerged, enucleation; if there are no symptoms, leave it alone. When a little bit is taken off the inner surface it was found that the child is brought back two or three years afterwards, the mother saying that the tonsil has grown again. Of course in these cases the tonsil had not grown; it had never been taken away. In most of the cases he referred to, the adenoids are really the important things to take away, and one simply, so to speak, took a little bit of tonsil in passing. And one is left with an uncomfortable feeling of having done a very inefficient operation.

Dr Elliott mentioned the snare, personally he had not tried the snare. He began this operation two years ago in fear and trembling, and the ease with which the tonsil came out in his first case encouraged him to go on, and he had found the operation a comparatively easy one. One dealt with the tonsil as one would with lymphatic glands elsewhere; one would not take a bit out if one could possibly take the whole gland away. He was quite prepared to try the snare, but in order to put it on it was necessary to separate the pillars and also to release the velar lobe. What Dr Porter said was quite right; the whole operation depends on getting into the proper plane.

Dr Lithgow mentioned the punch. He (Dr Fraser) bought a punch five or six years ago in Vienna, and he would be quite glad to hand it over to Dr Lithgow; he had tried it without any success: section is better.

He was obliged to Mr Struthers for what he said about the local anæsthesia and the advisability of giving a weaker solution.

Dr Dowden spoke about hæmorrhage. He (Dr Fraser) had had two bad cases of hæmorrhage after removal of the tonsil, but both of these cases yielded to pressure. Peroxide of hydrogen and turpentine, and pressing in upon the tonsil are usually sufficient. The external carotid ought to be ligatured, if any ligature is called for.

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2. ON THE DIAGNOSIS OF URINARY CALCULI BY MEANS OF THE X-RAY

With Lantern Demonstration

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During the past three years 143 cases of possible urinary calculus have been sent to me for examination. In 102 of these a negative diagnosis was made, and as showing the value and reliability of even a negative diagnosis, it may interest my readers to know that five of the patients suffered from severe and even dangerous hæmaturia, accompanied by persistent or intermittent pain. One proved to be a case of hæmophilia. The second, a man 31 years of age, died from what turned out to be a rapidly growing malignant tumour of the left kidney. The third patient died suddenly in hospital from cerebral hæmorrhage, and one may safely infer that he also had hæmorrhages into his kidneys which accounted for the hæmaturia. In the case of the fourth patient the kidney was incised, and a bougie passed down the ureter into the bladder ; no stone was found. The patient made a good recovery, and the hæmaturia ceased. In the fifth case the kidney was removed and a simple papillomatous tumour was found growing from the inner wall of the pelvis. Here, too, the patient made a good recovery, and the hæmaturia ceased.

Included in these negative cases are a number in which, owing to the presence of other conditions which cast shadows closely resembling those of urinary calculus, a definite diagnosis could not be made by the X-ray. These I shall return to later.

Of the forty-one cases in which a positive diagnosis was made, in thirteen of them no operation, so far as I am aware, has as yet been performed. In five—four of ureteric and one of vesical calculus—the stones were passed *per viam naturalem*, leaving twenty-three cases which went on to operation ; in two of these no stone was found.

The cases I have enumerated resolve themselves into three series, viz. :—Those in which an absolute *negative* diagnosis was

made ; secondly, those in which an absolute *positive* diagnosis was made ; and thirdly, those in which, owing to the character of the shadow, it was found impossible to say *definitely* whether it was due to a calculus or to one of the other conditions, the shadows of which mimic those of urinary calculus, the conditions most frequently giving rise to this difficulty being *phleboliths* or calcareous deposits in the pelvic veins, mesenteric *glands* which have undergone calcareous degeneration, and abnormalities connected with the *appendix*.

In order that the opinion given by a radiologist may be reliable, certain rules of procedure must be closely followed—rules which have been laid down by workers who have had a far wider experience than has, so far, fallen to my lot.

Needless to say, the technique must be good, and result in skiagrams which reveal more than an indefinite shadow of the parts exposed. The tissue markings of the 12th rib and of the transverse processes of the lumbar vertebræ, the edge of the psoas muscle, and even the shadow of the kidney itself, must be clearly discernible.

In every case of suspected urinary calculus a complete examination of the whole urinary tract on both sides must be made. Fig 1 shows the shadow of a calculus in the left kidney of a lady who suffered from hæmaturia, and in whom the pain was entirely confined to the *right* side. She came to me direct from the consultant's house. The large intestine had not been emptied. I made a complete examination of both sides, and found the stone on the left side, but nothing on the right. This condition, whilst occasionally met with, is rare. Following my advice she returned after the lapse of a month. The bowels had been thoroughly emptied, and I was able to demonstrate the presence of a calculus in the right kidney (Fig. 2) as well as the one in the left, which had been entirely unsuspected.

A patient was sent to me with the provisional diagnosis of calculus in the *left* ureter. Her right kidney had been incised, fragments of a large calculus had been removed from an abscess cavity, the kidney had been drained, and was out of circuit. I made a complete examination, and, as Fig 3 and Fig. 4 show, there still remained a fragment of a stone in the right kidney, and there was also present a large calculus in the bladder. The left kidney and ureter were empty. I should like at this point to draw the attention of surgeons to a question which has been put to me more than once, viz. : If it is possible to make an absolute positive or

negative diagnosis in the case of vesical calculus by means of the X-ray, why have patients to submit to the "brutality" of having a sound passed into the bladder? I need hardly add that in the case I have illustrated—Fig. 4—a sound had not been passed.

Fig. 5, a large round shadow in the neighbourhood of the spine of the ischium on the left side, is from a patient who had suffered for three years from symptoms of urinary calculus. For three months there had been a fixed pain about two inches internal to the left anterior superior iliac spine. The case was sent in as one of calculus of the left ureter. The tube was centred over the painful spot, with the result shown in Fig. 5, but a complete examination having been made, a large calculus (Fig. 6) was found to be present in the left kidney.

The operation was performed two days after the exposure to the X-ray had been made. A calculus, corresponding in size to the shadow, was found in the kidney. A bougie was passed down the ureter and that organ was found to be *empty*.

It may be that the shadow in the ureter region is that of a single phlebolith, or it may be that in the interval that elapsed between the taking of the skiagram and the performance of the operation the calculus had passed on into the bladder.

When dealing with *ureteric calculi* it is of the utmost importance that a control examination be made on the morning of the operation for their removal, because these bodies are liable to travel considerable distances at irregular intervals of time, and it has frequently occurred that an operator has found no stone at the point indicated by the skiagram; and, indeed, as has been pointed out by Dr Thurstan Holland of Liverpool and Dr Dawson Turner of Edinburgh, a calculus may travel up and down the whole length of a dilated ureter. Fig. 7 shows a calculus in the left ureter lying across the transverse process of the second lumbar vertebra. A control skiagram was made a few hours before the operation and it was then found to be (Fig. 8) lying below the level of the transverse process of the third lumbar vetebra, a position much more favourable for its removal, as was found to be the case by Mr Harold J. Stiles, who performed the operation.

Renal calculi, which give rise to severe pain and hæmaturia, are frequently small, flat and sharp-edged, and it is impossible for the surgeon to recognise their presence by mere palpation of the kidney. In such cases that organ must be incised. Fig. 9 is rather a poor print from the skiagram of such a case. Mr Stiles, who

performed the operation, was quite unable to feel the stone by palpation—in fact he expressed the opinion that it would have been well had a control examination been made that morning, inferring that the shadow was that of a ureteric calculus. Immediately on making an incision, however, and introducing his finger into the kidney wound, he came upon, and removed, a small, flat, sharp-edged stone about the size and thickness of the nail of the little finger.

Turning next to that very important and troublesome series of cases, in which it is difficult or impossible for the radiologist to give a definite opinion, we find that the most frequent cause of difficulty is the presence of *phleboliths* or thrombi in the pelvic veins which have undergone calcification, and which throw shadows closely resembling those of calculi in the lower third of the ureter. When these shadows are multiple, as in Fig. 10, it is comparatively easy to differentiate between them and those of ureteric calculus, though in the case illustrated Mr Thurstan Holland—whose repeated acts of kindness and guidance I gratefully and gladly acknowledge—to whom the plate was submitted, was of opinion that probably all the shadows were phleboliths, but that the top-most one *might* be a ureteric calculus.

The difficulty is greatly increased when only a single shadow is found, as in Fig. 11. This is from a young man who suffered for some months from pain of an indefinite character on the right side of the abdomen. The symptoms were not characteristic of appendicitis, and there was no blood in the urine. A skiagram was taken on three different occasions, and the shadow was found to be always in the same position. A guarded negative diagnosis was given. On performing an exploratory laparotomy, Mr Stiles found a small malignant adenoma growing from the tip of the appendix. On the patient's recovery from the operation a fourth skiagram was taken, and the shadow was found to be in its original position, and was evidently produced by a phlebolith.

Fig. 12 is from a patient, a woman, who had suffered for months from pain of a colicky nature, confined to the left side of her abdomen. She had, in addition, a considerable number of red blood-cells in the urine. After the first examination a guarded negative diagnosis was made. She was sent home, but returned in six months with the pain unabated, and with red blood-cells still present in the urine. A second skiagram showed the shadow to be present in its original position. As the patient was suffering

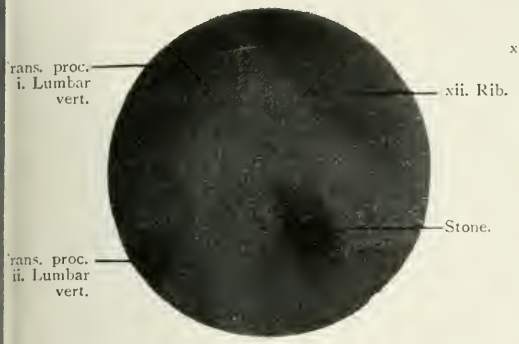

FIG. 1.

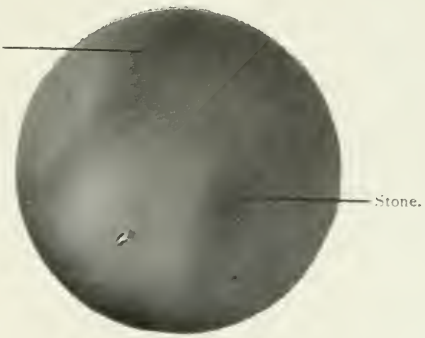

FIG. 2.

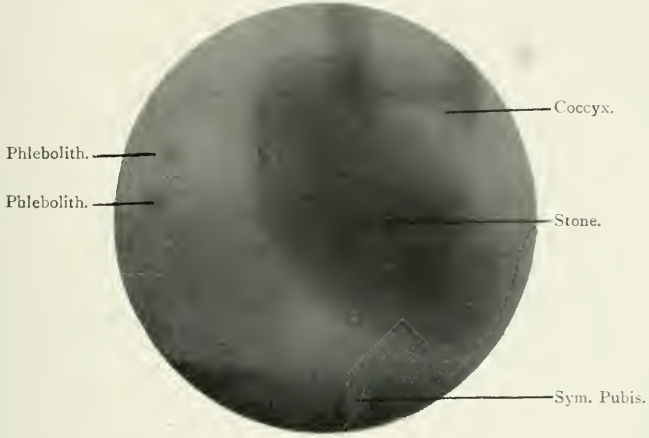

FIG. 4.

FIG. 3.

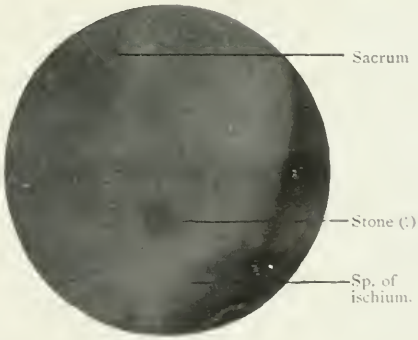

FIG. 5.

PLATE XI.—TO ILLUSTRATE MR EDMUND PRICE'S ARTICLE.

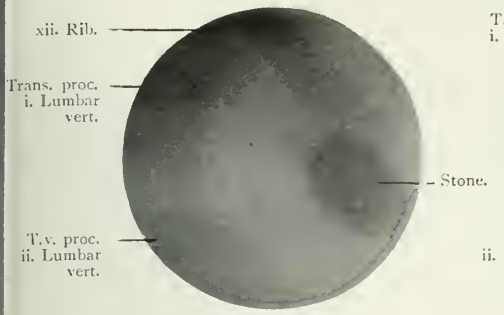

FIG. 6.

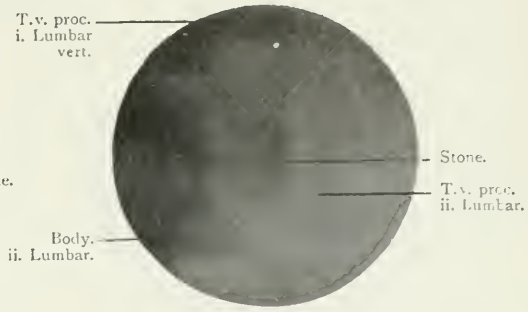

FIG. 7.

FIG. 10.

FIG. 8.

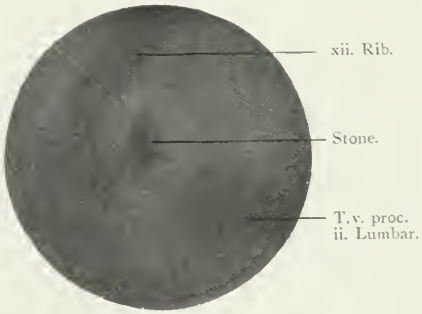

FIG. 9.

PLATE XII.—TO ILLUSTRATE MR EDMUND PRICE'S ARTICLE.

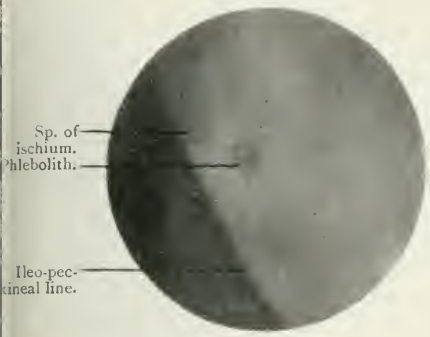

FIG. 11.

FIG. 12.

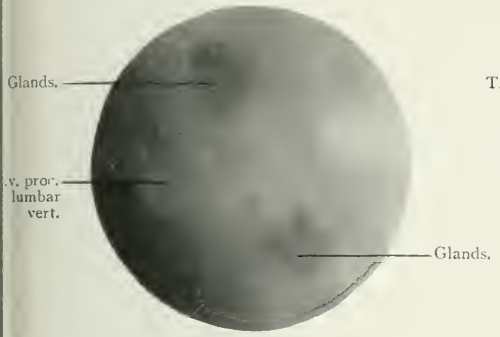

FIG. 13.

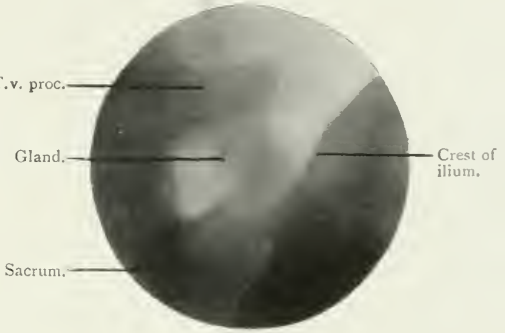

FIG. 14.

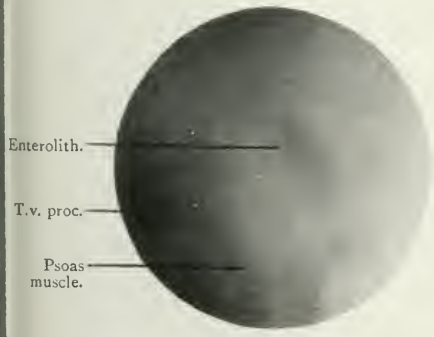

FIG. 15.

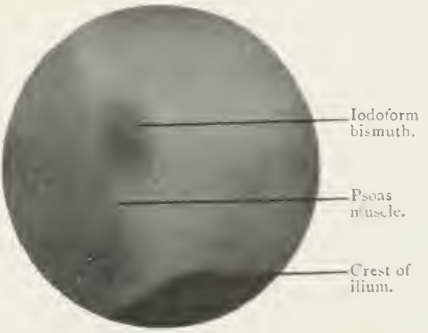

FIG. 16.

PLATE XIII.—TO ILLUSTRATE MR EDMUND PRICE'S ARTICLE.

a great deal, and was anxious to have something done, the ureter was cut down upon and found to be empty, but about half an inch external to it a small phlebolith was found and removed. The patient made a good recovery, and returned home completely relieved of the pain and hæmaturia.

Glands in the mesentery which have undergone calcification sometimes give rise to difficulty, not, as shown in Fig. 13, when the shadows are arranged in packets, as pointed out by Mr Hurry Fenwick, but when, as in Fig. 14, a single shadow is thrown in the direct line of the ureter.

Finally, abnormal conditions of the appendix are apt to cast shadows, which give rise to confusion and doubt.

Fig. 15 is from a lady whose symptoms had baffled a considerable number of medical men. The shadow, taken by itself, might quite well have been mistaken for a calculus in the lower pole of the kidney, but, fortunately for myself, I had made a complete examination, and was able to show two small shadows on a level with, and a little external to, the transverse process of the first lumbar vertebra, and the diagnosis of calculus in the kidney with an intestinal concretion in the appendix was verified by Professor Alexis Thomson, who performed the operation.

Fig. 16 is from a lady who had had her appendix removed two years previous to being sent to me for radiographic examination. The shadow is seen to lie in an unusual position and does not correspond with the normal line of the ureter. The conclusion I came to, and believe to be the correct one, is that it is thrown by the bismuth and iodoform paste which is usually applied to the stump of the appendix before the latter is invaginated and the peritoneal coat stitched over it.

In conclusion, I regret that some of the radiographs are not as clear as I should have wished them to be; they appear as they came from the printer's frame, and much detail is unavoidably lost in printing.

Discussion

Dr Dawson Turner remarked on the rapidity and extent of the change of position of calculi, and mentioned the case of a lady in whom the X-rays showed a calculus in the upper part of the ureter. The patient got up and changed her position and then another photograph was taken which showed a calculus in the lower part. This calculus presented similar appearances to the one seen in the upper

part, and on re-examination the one in the upper part was found to have disappeared; evidently within half an hour there had been this great change in position.

With regard to the question, How is one to know that the shadow is a calculus? he said there were three positions in which a shadow might be expected to be a calculus, namely, close to the kidney, in the lower part of the ureter, and just passing over the brim.

3. PERSONAL EXPERIENCES IN THE USE OF SALVARSAN "606"

By J. W. Dowden, F.R.C.S.

During the year lately ended there have been treated in the Lock Wards of the Edinburgh Royal Infirmary 586 cases of venereal disease, and of these, 217 suffered from syphilis. When the occupations followed by those infected people, mainly in the secondary stage of the disease, include those of butchers, bakers, dairy workers, fishmongers, cooks, car conductors, waiters, chocolate packers, laundry maids, etc., it becomes necessary to the community to cure them as rapidly as possible of all contaminating lesions. It is, however, a matter of interest that, notwithstanding such widespread disease, extragenital infection is rare, and there is still another matter of interest, and that is, the infrequency with which tertiary syphilis occurs when such large numbers of people are infected.

Those who have considered Ehrlich's epoch-making work cannot help being struck by his masterly study, for he has scientifically produced, after much labour, thought and experiment, the most potent drug as yet found in the treatment of syphilis.

During the past four months some 40 cases have been treated with salvarsan, mainly on account of the severity of the manifestation as well as the nature of their occupation.

The dose usually administered is .6 grammes, or, roughly, 9 grains of the drug, and this has been given by the intravenous method. Only twice has the injection been administered intramuscularly, but on account of unpleasant local effects was discontinued.

The method of intravenous injection which has been adopted is as follows:—

The patient is admitted the night before the injection. The various systems are examined, and permission obtained from

the parents if the person is a minor. The preparation adopted is that for a general anæsthetic—a purge the day before, practically no food except liquid on the morning of the transfusion.

Locally, the bend of the elbow and adjacent regions are prepared at the time by ether and tincture of iodine. An elastic webbing bandage is applied high on the upper arm, and the patient is directed to vigorously open and close the hand. The median basilic vein usually stands out most prominently, but variations in the size of the vein are striking.

The apparatus employed is that used for ordinary transfusion of saline, and in addition a hypodermic syringe with a fine needle, three pairs of artery forceps, dissecting forceps, dissector and knife are all boiled. Catgut, needle and horse-hair are also prepared.

In the Infirmary the salvarsan solution is prepared according to the directions attached to each tube of the drug, and the preparation is sent up in a sterilized flask. In another flask there is a pint or more of normal saline solution. These two flasks are placed in warm water half an hour before use. The salvarsan solution should be clear, yellow, somewhat like picric acid solution, and if cloudy, ought not to be used till, by the addition of a drop or two of a fifteen per cent. solution of sodium hydrate, it is rendered clear. In private work a chemist would prepare the solutions and send them ready for immediate use.

When all is ready saline solution is poured into the glass funnel and allowed to run freely through the needle, then the rubber tube is clamped. If the vein is large and prominent, as is usually seen in the male, the needle is driven into it at an angle of 45°, and it is known whether the vein is pierced by the forcible spurting of venous blood on each side of the needle. Once in, the needle is gently turned in the line of the vein, lest the walls be re-perforated. The elastic webbing bandage is now removed from the upper arm and also the clamp on the rubber tube, and saline is allowed to run into the vein, which, as a rule, it does readily, but if it only passes slowly from the tube, with swelling of the tissues, leakage has occurred and absolutely no salvarsan should be transfused. If the needle is correctly in position, the rest is easy. Saline should be allowed to flow until just disappearing in the rubber tubing, which is then clamped and the salvarsan solution now poured into the glass funnel. Each time salvarsan solution is added to the funnel, it is as well to momentarily clamp the rubber tubing with the fingers, lest air bubbles gain access. When the last of the solution has just

entered the rubber tubing, a clamp should be applied and saline now poured into the glass funnel and allowed to wash the last of the salvarsan solution into the vein, while in addition, the escape of salvarsan into the tissues is avoided. Salvarsan transfused into the tissues is most deleterious locally. The disadvantage of "plunging" for the vein is that, if unsuccessful, there is troublesome venous bleeding which annoyingly interferes with the exposure of the vein. If the vein is small or plunging has failed, the vessel must be exposed under cocaine anæsthesia and the cannula directly inserted into the orifice.

It need hardly be mentioned that it should not be forgotten to remove the elastic webbing from the upper arm.

After transfusion has been carried out, by the open method, the vein above and below is tied and a mattress suture of horse-hair is inserted as the edges tend to become inverted.

Gauze and wool are applied to the bent arm when the patient goes back to bed, and nothing is given by the mouth for several hours.

In a couple of hours, as a rule, there is reaction—a feeling of heat, and with it there is a rise of temperature to 99° F. or 103° F., some squeamishness or actual sickness. The reaction seems most marked in imperfectly prepared patients and in men. Women show a much milder reaction. The symptoms settle soon and disappear, as a rule, in twenty-four hours.

As regards the syphilitic lesions, occasionally definite changes are seen in twenty-four hours, mainly, however, as a reaction of a hyperæmic nature. The primary sore looks healthier and bleeds readily. In one case .3 grammes was given to a patient whose primary manifestation had healed under mercury and who had no secondaries, with the result that the scar broke down and a typical hard sore appeared. He was then given a dose of .6 grammes, when healing rapidly took place. Secondary lesions of the skin gradually fade after some initial exacerbation, but in papular rashes there is a tendency for a pigmented spot to mark the site of the lesion. In one case, where the eruption was almost rupial in character, the reaction of the drug was very slow. The temperature was nightly 99° F., the same as before the use of salvarsan. At the end of a week mercurial inunction was employed, with the result that the temperature fell to 98° F. in twenty-four hours, and improvement promptly took place.

Condylomata clear up, not with extreme rapidity or even more

rapidly than with inunction and careful local attention. Hair ceases to fall out in a week or so. The lesions that react most promptly to the drug are the secondary conditions about the throat, mouth and tongue, often most obstinate and troublesome to heal, and especially those secondary manifestations about the soft palate, pharynx and tonsils in which there is extensive and rapid loss of tissue, resisting ordinary local and general treatment.

In the late secondaries, or early tertiaries, remarkable results are seen, especially where the disease has steadily resisted all other treatment—multiple gummata of the skin, ulcerations of the palate and face of a lupoid type. I shall give one case as an example. A man, aged 30, with a doubtful history of syphilis, had for three and a half years been troubled with a disfiguring hypertrophic and ulcerating condition about the nose. He also had ulceration about the soft palate. All forms of treatment had been tried—mercury, arsenic, pot. iodid. He reacted to bovine and human tuberculin, was treated with tuberculin ointment and X-rays, but steadily got worse. He was only too willing to have any experiment tried, and the injection of salvarsan .6 grammes was given intramuscularly in the buttock. There was a local reaction of very intense pain, which required morphia, and as regards the nose, it becomes redder and more swollen, but by the end of a week to ten days, the whole condition had become entirely cured.

As regards that most troublesome condition, old-standing leukoplakia of the tongue, salvarsan has practically no effect, nor could it be expected to have any as far as cure is concerned and the restoration of scar to normal tissue.

With regard to the general effects, the patients have all expressed themselves as being remarkably improved and possessing a general sense of well-being. They usually also put on weight. I have never seen any bad general results.

As far as local results are concerned, intramuscular injection has been associated in one case with intense pain, but slowly disappearing, leaving stiffness for a month or more afterwards. In another case a large swelling appeared, which ultimately ruptured, with the escape of a clear serous discharge, but this healed readily. Intravenously the bad local results have been where the tissues were infiltrated with some of the solution. There is marked local oedema, threatened suppuration, and in one case the skin over a small area became black and gangrenous.

There is no effect on gonorrhœa.

In one case of syphilis associated with scirrhus of the breast, in which a diagnosis of malignant disease had been made by microscopical examination, the injection of salvarsan made no difference in any way whatever.

Venereal warts are not affected by the drug, showing that some other variety of an infective nature produces them, as the hypertrophic syphilitic developments, such as condylomata, react and disappear.

Mercury is always given with "606," because a cure with salvarsan is as yet not quite certain. Although many writers state that two injections of salvarsan, at an interval of some seven to ten days, is sufficient to cure the disease, it is perhaps wise to err on the safe side and continue with mercury. This may cover my impressions so far as the curative effects of salvarsan go.

My general impressions with regard to salvarsan are, that in it we have the most potent and rapidly acting drug existent for the treatment of syphilis, but as to its ultimate curative effects time alone will show.

Discussion

The *President* said he was sure the members would agree with him that they were much indebted to Mr Dowden for bringing this very practical and important subject before the Society.

In connection with the discussion he would like to ask the opinion of the physicians and surgeons present with regard to a question in a letter which he had received from one of the physicians in Hull. "Is it the custom of your physicians to perform intravenous injections, either in the hospital or in private practice? Do you regard such a proceeding as 'practising surgery,' and is your opinion altered by the fact of it being necessary in many cases to make an incision through the skin and introduce a cannula into a vein?" It was an interesting question on which he hoped to hear an expression of opinion.

Dr T. J. Thyne asked Mr Dowden whether he had had any experience of giving two doses of .6 grammes of salvarsan. Recently he (Dr Thyne) had tried the drug in a case of pernicious anæmia and had given four doses of .3 grammes, with the result that the patient had well marked symptoms of arsenical poisoning. He had injected the remedy into the buttocks as had been suggested. A rash appeared all over the body, there was numbness in the arms,

and the patient was very ill; at the same time she derived enormous benefit so far as her anæmia was concerned, the corpuscles had gone up from 1,000,000 to about 2,000,000. He had also tried it in other three cases of a different kind, but there had not been the same severe reaction.

Mr Wade said he would like to relate a little of his and Mr Wilkie's experience of patients with syphilis treated by this drug. They had had 15 odd cases, and they comprised primary, secondary, and tertiary syphilis. Anyone who had seen the marvellous effects this drug produced must be impressed by the way in which it makes syphilitic lesions, whether primary, secondary or tertiary, disappear. Their very first case offered to them very hopeful prospects. The case was that of a married man who had contracted syphilis and who responded extremely badly to the usual remedies. He had been twice in the Lock Wards of the Infirmary, and through idiosyncrasy responded exceedingly badly to treatment. When first seen he was a most miserable object—depressed in spirits, thinking of suicide, studded all over with numerous ulcers, and suffering from persistent diarrhœa. At this time they had only had one day's opportunity of using the remedy, and after explaining to him that this was a new remedy of which they did not know very much but were prepared to give him the benefit of if he were prepared to take the risk, and telling him what the experience of other medical men had been, the patient consented to have it. After some eleven days six of the seven ulcers disappeared, and the seventh one healed by the middle of the third week. The experiment was conducted as well as any experiment could be, because in that case they treated the man as an outpatient, there was no difference in his environment, and the same filthy rag was fastened round his leg. He would not detail the other cases, but wished to say that the same astonishing improvement resulted. In all of the cases it apparently caused a certain degree of discomfort, but within a day or two that had passed away, and the patient looked comparatively happy. In all of them, in addition to the local improvement, there was a feeling of general well-being which seemed to continue. In all of the cases the intravenous method was employed, the drug being poured into the vein just as one would do with any other saline injection.

According to the literature it is considered the orthodox treatment to follow up the intravenous injection by injecting another

dose into the buttocks a fortnight later, the idea being that after you have destroyed the organisms you give this second injection with the object of destroying any that may have escaped in the general slaughter. They had not done that, the reason being chiefly the (10/3 a dose) cost; so far no recurrences of the condition had taken place. The patients are all doing well, and some of them have gone on for several months; if there were any signs of recurrence they would have resort to a second injection.

Dr Logan Turner said one was struck with the remarkable improvement which occurred in these cases, but there was a tendency at the introduction of a new drug to try and adapt it to the treatment of other conditions. Knowing the value of arsenic in lupus, he had attempted injections of salvarsan in four cases, but absolutely no improvement followed.

Mr Wilkie said there was one case which Mr Wade had not referred to which illustrates how the drug acts and what it is that causes the reaction. The case was that of a man with ulcers on the side of the tongue and floor of the mouth. There was a very definite history of syphilis some years previously, but the appearance was suspicious of epithelioma, consequently a small piece was removed and examined microscopically and was found simply to be inflammatory infiltration. The patient was put on iodide for two weeks and decided improvement resulted; at the end of that time an injection of salvarsan was given intravenously. There was not the least reaction of any sort—no rigor, headache or sickness, which are seen almost invariably after intravenous injection. A Wassermann was done the same day, but was negative. Further microscopic examination revealed the ulcer to be epitheliomatous. That suggests that the rigor and reaction are due to antitoxin from the spirochætes.

The question is, Is one injection going to be a cure? Results, however wonderful they may be at the time, do not justify a pronouncement that one injection will cure the condition. To demonstrate the spirochæte it is sometimes necessary to cut numerous sections of a syphilitic lesion; spirochætes are not found throughout the lesion, but in little bags round which are found small arteries and veins which show induration, consequently one dose of the drug cannot in every case be expected to reach the spirochætes. That all the symptoms disappear is not proof that the disease is absolutely

cured, because years afterwards these spirochætes may be liberated and cause further symptoms. In one of their cases, where the man had indurated tissues, he was advised to foment the part for twenty-four hours after the injection in order that the drug might be better transfused, he had not yet heard the result.

Dr Chalmers Watson asked Dr Thyne what the extent of the local reaction was in the case of pernicious anæmia to which he had referred. At present he (Dr Watson) had a case of pernicious anæmia under his care in which the results so far had been somewhat disappointing. The interesting thing was the enormous local reaction. In the buttocks there was an enormous tumour, almost the size of a fœtal head, which persisted for many weeks. As regards the blood there was no improvement, in fact deterioration. He had given up the drug and gone back to the old arsenic preparation, which up to the present had produced notable improvement.

Dr T. J. Thyne said it was now ten weeks since the first injection. On the buttock there was a lump about the size of a hen's egg, which was tender and became discoloured; that has gradually disappeared, but has left a considerable inflammatory mass. The patient had been previously three times in the Infirmary, and had at first improved immensely under arsenic, but, as so often happened in pernicious anæmia, the second and third times there was little or no improvement. Salvarsan had no doubt saved her life so far; she had been confined to bed but was now able to be up.

Mr Scot-Skirving said Mr Dowden had referred to the comparative painlessness of intravenous injections. He was greatly struck when in Berlin last summer—he did not see Exner who was away at the time—with the statement that there was little pain. He understood that Exner was in the habit of giving the drug almost entirely subcutaneously, and said there was very little pain. He had questioned some of the patients and found they absolutely confirmed that point; although the drug had been given subcutaneously the majority of them said they felt little pain at all. It is said that Exner did not get the pain that others did. Mr Dowden said it was the acidity which caused the pain; perhaps if the solution was made absolutely neutral there might be no pain.

Then Mr Dowden found that in the primary sores one did not

get the marvellous results which are seen in the secondary lesions; that seemed to corroborate Mr Wilkie's suggestion that you get a great deal of indurated tissue in an early chancre, and that the induration prevents the arsenic getting at the spirochætes which are, of course, more located in the chancre.

Although one did not want to bother the reader of the paper with questions, he would ask about blindness. The bugbear of all the early arsenic preparations was the danger of blindness. He had taken a pretty keen interest about two months ago in the subject of salvarsan, and at that time no cases of blindness had occurred as the result of salvarsan. He would be glad to know how many cases of blindness had been recorded in the literature?

Mr Dowden alluded to the presence of a flocculent precipitate and the danger of it. There was not very much danger. In one case about a month ago he had administered salvarsan which was in a fairly flocculent condition and no untoward result followed; the patient ate a very hearty meal within two hours of the injection and was not the least sick.

The *President* said he was sorry no one had answered the question he put as to the intravenous injection of salvarsan by physicians. Speaking for himself he did not see why physicians should not inject this remedy and make incisions for the purpose of doing so. Physicians were in the habit of performing surgical operations far more difficult than this—lumbar puncture, bleeding, the latter not often now, unfortunately. For his own part he claimed that physicians are quite capable, if they choose to do so, of practising this method, and that was the reply he proposed to send to his correspondent. He should have liked to have had an expression of opinion from some of the members.

He had been very much interested in Mr Dowden's paper, but he had not referred to one important condition—general paralysis of the insane. He believed Mr Dowden had had considerable experience in Morningside with regard to G.P.I., and he (the President) had been told that some good results have followed in these cases. The parasyphilitic lesions, such as tabes and G.P.I., he supposed, were very much on the same plain as leukoplakia, and one could hardly expect with a remedy of this sort to get very much benefit; the practical question is whether it really does good or not. To-day he had, as it happened, been consulted by a patient whom he had seen off and on for the last sixteen years with syphilis, who was

suffering from leukoplakia—he had written to him some time ago telling him of this remedy, and the patient, being in town, came to see him. Mr Dowden seemed to think from his experience that it is not likely to do much good in leukoplakia. Dr Bramwell had told the patient he had been very much impressed by the beneficial results in many cases, and that if he had his condition of the tongue he should have an injection. That is the best answer one could give to a patient. If the remedy does not do any good, it does not do any harm, and there is a chance of its doing some good. If he had syphilitic leukoplakia he should like to have an injection, he did not mean to say that he should like to have it frequently repeated, but he would like to see what improvement one injection might produce. If he had tabes or G.P.I., he certainly would like to have more than one injection. In two cases of pernicious anæmia, which he had published some time ago, and which had attracted a great deal of attention, the improvement was certainly most remarkable, and he was glad to hear that Dr Thyne's case has corroborated them. Of course one could not judge from one or two cases, but the improvement was very remarkable. There was very great reaction in both of these cases, and he believed the explanation was the one just suggested by Mr Scot-Skirving, that the saline should be absolutely neutral otherwise you get a marked reaction. There was another case of pernicious anæmia in his ward in the Infirmary which had been treated while he was away, and in it also very great improvement has taken place. The drug, of course, is in an experimental stage, but he did hope that in some diseases in which arsenic is beneficial improvement would result, and it seemed to him to open up a very large question with regard to the treatment of many diseases. If they could get a drug which could cure the germ of syphilis, as salvarsan undoubtedly does, and produce such enormous improvement, might they not expect to find some drug which will attack other germs, and which would be infinitely better than the vaccine and other plans of treatment. Although salvarsan might not cure tabes and G.P.I., yet there is every hope that it will prevent these diseases.

Mr Dowden said, in the first place, he would like to thank the members for listening so patiently to his remarks. He might, in passing, answer the question as to whether the physician or the surgeon should administer salvarsan by saying "it is not always so easy as it seems."

They had already learned from Dr Chalmers Watson and Dr Thyne that there was great local reaction. With regard to repeated doses, he had had two cases in which he had given a second dose, and in one of these the man was tested with the Wassermann reaction later. The injections were given at intervals of six weeks, and the Wassermann was negative. But knowing the disease, syphilis, he did not take it for granted that salvarsan is a cure, and therefore carried on the treatment with mercury at the same time. Dr Thyne, he thought, had found in every case a rise of temperature. There is always some reaction—a feeling of heat, marked temperature, sometimes rigor.

Mr Wade spoke of the treatment of these cases as out-patients. He (Mr Dowden) had not had the honour of being asked in private to carry out this treatment, but in an Institution like the Infirmary it was, he thought, very important indeed that all patients should be admitted in case some untoward result might happen, and the Institution have the " benefit " of the newspapers. He admitted every patient as a rule, unless he goes back in a cab to his home and is under the care of a qualified man. In the case of minors he got the parents' written consent before he performed this operation, because of the discredit which might befall the Institution should any accident occur.

In intramuscular injection the arsenic is stored up for a long time and the organs become accustomed to the effect of the small dose, consequently the bigger dose would not have the same effect. The whole idea is to kill the germs off " at one shot."

With regard to Mr Wilkie, he should have mentioned that the drug is useful in other things besides syphilis. It has no effect on gonorrhœa, as he had found in his department.

Notwithstanding Mr Scot-Skirving's remarks regarding the statements of the Germans that there was little or no pain, all the people he (Mr Dowden) had seen had complained of very great pain. He had not kept a record of his cases and could not say how many he had had. Mr Scot-Skirving must be congratulated on his courage in injecting a patient with a flocculent solution. As far as G.P.I. is concerned he (Mr Dowden) had no experience.

Meeting VIII.—June 7, 1911

Dr Byrom Bramwell, *President, in the Chair*

I. Exhibition of Patients

1. *Dr Edwin Bramwell* showed—

A case of PSEUDOHYPERTROPHIC PARALYSIS.

Male, aged 12½, the eldest of five; others all healthy. No known case in family. Always a bad walker, could never run. Typical gait. Great enlargement of calf muscle and intraspineti, slight of masseters and (?) tongue, etc. Latissimus dorsi and lower part of pectoralis major practically absent, etc.

2. *Mr Scot Skirving* showed—

Man, æt. 54, after operation for PERFORATED PYLORIC ULCER.

Shown (*a*) as an example of "silent" ulcer of the pylorus, no symptoms having been present before rupture, although operation revealed a chronic ulcer of large size.

(*b*) Owing to concomitant heart disease, no general anæsthetic was used, and the operation (which necessitated a gastro-enterostomy) was conducted entirely under novocain infiltration.

3. *Mr J. W. Struthers* showed—

Patient showing result after excision of astragalus for pneumococcal arthritis of ankle joint.

4. *Mr Charles W. Cathcart* showed—

RUPTURE OF THE SPLEEN—LAPAROTOMY—RECOVERY.

William C., aged 18, carter, was admitted to ward 18, Royal Infirmary, on the 16th of May 1911, suffering from shock and abdominal pain caused by a severe bruise.

He had been walking beside his cart, which was heavily laden, when the horse suddenly shied and caused the cart to swerve towards him. It knocked him down and the wheel went partly over the back of his chest.

When admitted, he was in a dazed condition, but could answer questions sensibly. Pulse soft, temperature 99°.

There were marks of bruising on the chest near the angle of the left scapula and on his right arm. No bones were broken.

He complained of pain over the upper part of the abdomen. The respiratory movements of the abdomen were restrained and the abdominal muscles were tender on pressure and felt rigid.

About an hour after his admission he vomited about 8 ounces of coffee-ground material; and again, half an hour later, about 4 ounces of similar material.

Fearing that there might be a rupture of the stomach, Mr Cathcart opened the abdomen in the middle line about two hours after admission. The stomach showed no sign of rupture or even bruising, but extravasated blood was found in the left cupola of the diaphragm which seemed to come from the spleen. The left rectus muscle was therefore cut across; the wound was retracted. When the spleen was drawn forward, it was seen to be split in several directions as if it had been burst by pressure. Oosing was still going on from one spot. An effort was made to stop the oosing by pressure with a catgut stitch, but the tissue was so friable that the thread cut its way out at once. Steady pressure was then applied with a gauze swab wrung out of hot saline solution (about 115° F.), and in a few minutes the oozing ceased.

The abdominal wall was closed with chronicised catgut stitches and the skin with horse-hair.

The patient made an uninterrupted recovery.

Remarks.—The hæmatemesis must have been due to bruising of the gastric mucous membrane by the same compression which burst the spleen. And it is noteworthy that although this compression must have acted through the chest wall none of the ribs were broken. Had it not been for the hæmatemesis, the exploratory operation would probably not have been undertaken and the injury to the spleen would not have been recognised.

II, Exhibition of Specimens

Dr Chalmers Watson exhibited—

(*a*) Liver and spleen from a rare form of acute leukæmia. (In conjunction with Dr Miller.)

(*b*) Brain showing cerebral hæmorrhage from a boy in whom the clinical features closely simulated tuberculous meningitis. (In conjunction with Dr Drennan.)

III. Demonstration

Dr Chalmers Watson showed a series of naked eye slides illustrating the influence of a rich nitrogenous (purin containing) diet on the thyroid gland. The demonstration consisted of a series of five glands—the larger gland—taken from five animals fed on the special diet, cut serially from end to end, compared with an equal number of glands taken from animals of similar age and weight, fed on bread and milk. In every instance the glands from the animals fed on the special diet were much enlarged, the glands being of considerably larger size, containing double the number of sections. Histologically the glands of the animals fed on the special diet were more cellular than the control.

IV. Original Communications

1. INTESTINAL OBSTRUCTION AND A RECORD OF ONE YEAR—1910

By Rutherford Morison
Professor of Surgery, Durham University

When your Secretary honoured me with an invitation to read a paper before you it appeared to me impossible to find a more interesting subject than that of intestinal obstruction, for intestinal obstruction still remains one of the abdominal diseases in which surgical progress has made slowest advance, and the reasons are not far to seek. The first probably is that cases are relatively few, and that consequently each individual surgeon requires a long experience and large opportunities before his personal knowledge allows him to arrive at any helpful conclusions; the second that diagnosis is difficult; and the third that uncertainty in diagnosis leads to want of promptitude in action, which in these cases is disastrous in its results.

Notwithstanding all these difficulties some advances have been made, and to those I intend to draw special attention.

For some years I have taught that the diagnosis of intestinal obstruction is based upon a trinity of signs, viz. (1) Spasmodic pain; (2) Inability to pass flatus; and (3) Evidence of increased peristalsis. This symptomatology is based chiefly upon experimental evidence. In the performance of inguinal colostomy, except

in cases of emergency, it has been my practice to do the operation in two stages. The first comprises drawing a knuckle of the sigmoid colon through a small opening in the left inguinal region and leaving it there to form adhesions. During the 5 or 6 days which are allowed to elapse the patients are so comfortable that they make no serious complaint. The second stage is accomplished by tying an elastic ligature very tightly round the projecting intestine. The object of this is to avoid the troublesome and sometimes serious bleeding which may follow division of the bowel either by the knife or cautery. The application of the ligature causes no discomfort, and as a rule nothing is felt for some hours. Then " wind " begins to cause painful rumbling, and the patient frequently perceives that it is arrested at the ligatured spot. If nothing be done at this time all the symptoms of acute obstruction develop in some cases. In all, entire relief is obtained as soon as flatus escapes if a small tube be introduced through an opening in the intestine above the ligature.

With these symptoms and signs the diagnosis of intestinal obstruction due to a mechanical block, for which immediate operation is indicated, is the only sound one, and I almost regret to have to record an exception to this lest anyone should feel inclined by a knowledge of the possibility to counsel delay.

Case I.—*Paralytic Ileus.*—M. A., aged 35, married woman, was admitted to the Royal Victoria Infirmary, Newcastle-upon-Tyne, on 15/8/10.

She was sent in by her doctor as a case of ruptured gastric ulcer, and I saw her on admission.

There appeared to me to be insufficient evidence upon which to base a diagnosis, and I ordered some palliative treatment and a careful hourly note to be made of her condition. By the following day she appeared to be nearly well, and nothing of a seriously suggestive character remained.

Two days later (17/8/10) my colleague, Mr Richardson, saw her by request of the house surgeon at 10 o'clock in the morning. He then made a definite diagnosis of intestinal obstruction, but decided, as she had no urgent symptoms, that she should await my arrival. When I saw her at 12 o'clock she was shouting with pain. She had passed no flatus since the attack commenced 8 hours before, though wind belching had been frequent, and on exposing her

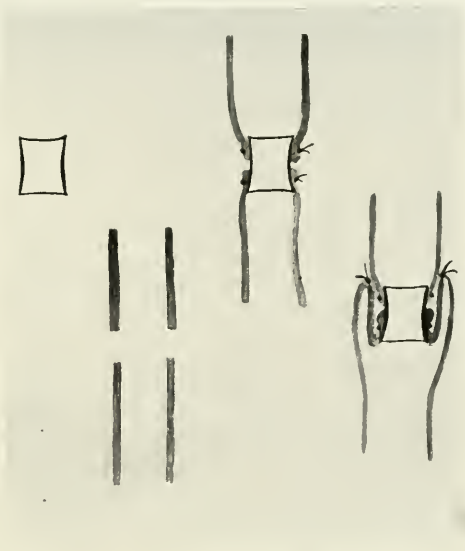

FIG. 1.—Glass Anastomosis Bobbin.

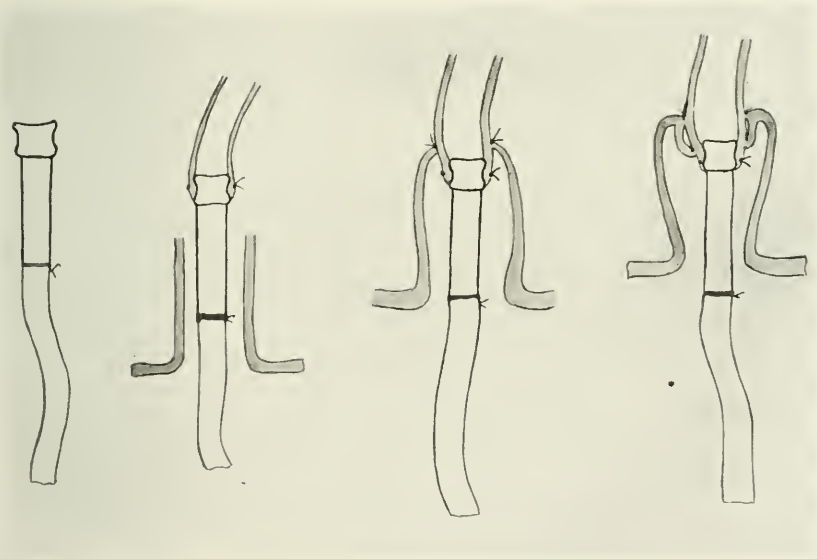

FIG. 2.—Glass Anastomosis Bobbin with India-Rubber Tube.

Steps of Operation.

1. Tie Glass Bobbin and India-Rubber Tube into the upper end of the divided sigmoid.
2. Take hold of outer wall of lower bowel with four catch forceps half-an-inch from its cut end.
3. Pass a solid bougie with thick silk ligature tied to its end into the lower bowel from above and out through anus. The silk ligature is to draw through a bunch of iodoform gauze followed by the india-rubber tube.
4. Pull on the india-rubber tube till the glass bobbin is entirely within the lower bowel.
5. With the aid of the fixation forceps double the lower cut end downwards (invert) and fix it there with two rows of suture.

PLATE XIV.—TO ILLUSTRATE PROFESSOR RUTHERFORD MORISON'S ARTICLE.

abdomen violent peristaltic movements were plainly visible and the hardened contracting intestines could be readily felt. Moreover, there was a basin beside her bed containing about 2 quarts of dirty feculent smelling fluid, which she had recently vomited. Her stomach was washed out and her abdomen immediately opened. No organic obstruction was found. The whole colon was much distended, the small intestine contracted at parts, in others distended. The urine contained about $\frac{1}{6}$th of albumen. In many previous papers I have directed attention to the fact that organic intestinal obstruction can be simulated by some intestinal disturbance occurring in connection with affection of the kidney, and this condition has more recently been described as enterospasm.

That shock occurring in a case of acute obstruction usually means obstruction plus strangulation, will generally be acceded, but shock may be present without strangulation, and strangulation without shock.

In the cases I have observed of shock without strangulation there has always been free fluid in the abdomen and marked congestion of the intestine above the obstruction, similar to that found with a strangulation. The most probable conclusion is that the physical cause of shock in these cases is a profound disturbance of the balance between the somatic and splanchnic circulations. That the tendency to shock is largely an individual characteristic is made certain by some of my experiences. One of the most serious instances I have seen of shock following an operation occurred to a nervous young lady on whom I had operated by ligature for hæmorrhoids. One hour after the operation, which was unattended and followed by no loss of blood, she became so ill that for 3 hours her condition appeared to be desperate, and her recovery occupied some hours before it was complete enough to relieve our anxiety. This same patient blushed on the smallest provocation, and in other similar instances I have noted marked vasomotor instability.

It is well recognised that in children the most common course of intestinal obstruction is intussusception. In them the diagnostic symptoms and signs have now become common property, the only proper treatment, immediate operation, is universally recognised, and the results of operation are as good as those of strangulated hernia 20 years ago.

It is not so well recognised that spontaneous reduction fre-

quently occurs—much more frequently I think than the spontaneous reduction of a strangulated hernia. One little girl of 6 whom I saw in the country is one of the most striking examples of this. At my visit I found her suffering from intussusception and advised her immediate removal to the Infirmary. As on her arrival there we found that all her symptoms had vanished, and along with them the tumour, I refrained from operating and she promptly recovered. Her parents, exceptionally intelligent people, were told when she was sent home to return at once if there was any recurrence of her symptoms. Within one year she was sent back three times with recurrence of her symptoms, but was either well on her arrival, or became so shortly afterwards. The day after her last admission she was entirely free from symptoms, and I opened her abdomen with the object of preventing further attacks. The operation confirmed the diagnosis, for the ileum close to the ileo-cæcal valve showed obvious damage as of recent strangulation, and pleating the mesentery sufficed to prevent return of her seizures. Another point which must be emphasised is that whether the intussusception is acute or chronic depends upon whether the intussuscepted portion of gut is or is not firmly strangulated. The chronic variety may be called a disease of adults, and the acute, one of children, but there are exceptions to both statements.

Case .—A male child, aged 3½ years, a patient of Dr Burnell, was admitted to a private hospital on 7th March 1909.

History.—His illness was of 10 days' duration. It began with pain and vomiting after eating an orange. After 2 days the active symptoms ceased, but he occasionally vomited and had spasmodic pains coming on at intervals until the last 6 days, when there was no sickness. His bowels had been moved for the last 4 days, but for the first time since the night before admission blood was noticed in the stools. He had eaten little during the illness and had been maintained on fluid foods. From the first Dr Burnell had been able to feel a tumour under the upper part of the right rectus muscle, but his diagnosis of intussusception was rejected by an eminent physician who was called in consultation.

On admission the child looked ill but not dangerously so. His tongue was dry and covered with a white fur ; temperature, 98° F. ; pulse, 130.

His abdomen was slightly distended and a definite elongated tumour could be felt under the upper part of the left rectus muscle immediately below the costal margin. During the examination visible peristalsis and audible rumbling developed, and the tumour was felt to harden during an attack of pain.

I confirmed Dr Burnell's diagnosis of intussusception, and operated.

The abdomen was opened through the right rectus muscle over the tumour, when it was found that the end of the ileum and appendix and cæcum inverted into the ascending colon formed the tumour which had been felt. Reduction was readily effected, and except for some œdema the gut appeared to be undamaged. Recovery was uneventful.

In adults chronic intussusception is more common than in children, but it is rare, and the diagnosis is still more difficult. It has been said that the definite sausage-shaped tumour alternately hardening with the pain and relaxing in the interval is pathognomonic, but the following case proves that even this sign may mislead.

Case .—*Malignant Stricture of Ascending Colon simulating Chronic Intussusception.*—A widow, aged 81, was admitted to a private hospital, 10th November 1908.

History.—Two years before admission she had several sharp attacks of abdominal pain with wind rumblings. Little importance was attached to them and she soon got quite well. One year later she had a repetition of similar attacks. The rumblings were then loud and with them there came a visible swelling in the right lower abdomen. She was often sick with the attacks, but the vomited matter had no bad smell. Vomiting relieved her pain, and she soon felt all right after a good clearance. Then followed 10 months of freedom from trouble, but during the last 2 months her pain had returned and she had got thinner. Nothing wrong was noticed with the stools—no diarrhœa, blood, or slime.

On admission her general condition was fair. Temperature, 98.2° F. ; pulse, 86 ; arteries, elastic. On examining the abdomen at first nothing was seen or felt. During the examination some rumbling occurred and marked peristalsis became visible and palpable. A large sausage-shaped tumour then formed on the right

side, reaching from the pelvis below, filling the right flank, and extending up to the right costal margin. The tumour became quickly very hard, and above and below a rounded well-defined edge could be mapped out. It could be moved from side to side, not so freely from above downwards, and very slightly with respiration. After less than 1 minute the swelling disappeared with a gurgle and the abdomen was as before.

There were no signs of disease anywhere else. The diagnosis made was chronic intussusception.

Operation, 17th November 1908.—The abdomen was opened by an oblique incision commencing in the right iliac fossa and extending back towards the loin. The cæcum and ascending colon presented, and on the latter there was a characteristic annular malignant growth causing a marked stricture about 5 ins. from the end of the cæcum. The terminal 5 ins. of the ileum, the ascending colon, and the ileo-colic lymph area were excised, the divided end of the colon was sutured, and the distended reddened ileum was brought out of the wound and tied to a Paul's tube for temporary enterostomy.

Second operation, 30th December 1908.—The fæcal fistula was temporarily closed with sutures and the wound was reopened. A further portion of the ileum was removed and a lateral anastomosis made with the colon above its closed end. Good recovery. Death occurred 1 year later. There was no post-mortem examination.

The fact that intestinal growths usually accompany adult intussusception is illustrated by the relation of my cases.

The next condition of which I wish to say something is *volvulus*.

It is a very important one, one quite frequently overlooked, and one concerning which there is much loose writing and thought.

In my recent book (*An Introduction to Surgery*) I have divided volvulus into two varieties, primary and secondary. The following is a good example of one of the primary forms :—

Case .—*Complete Volvulus—Four Operations—Radical Cure.*—A miner, aged 15, was admitted to the Royal Victoria Infirmary, Newcastle, on 21/10/09.

History.—Three times previously he had been in the institution—1/2/08, the 18/3/08, the 14/10/08—and had been operated upon for acute intestinal obstruction due to a complete volvulus. The notes

show that on each occasion it had been necessary to eviscerate him to reduce the twist, and the distended cæcum was always found near the spleen.

On the last occasion (30th October 1909), after reducing the volvulus, I attached his cæcum in the right iliac fossa and his ascending colon by a series of sutures in the right flank. Each suture of silk passed through the outer coats of the bowel and out through the parietes and was fastened there over a dossil of gauze.

4/6/11.—Patient has had no attack since the last operation, and is "stronger and better than ever before."

The secondary form is illustrated by cases in an earlier series. They demonstrate a fact not sufficiently recognised, but true of all twists, that it takes several attacks and often many twists to cause gangrene and death.

Colon.—The diagnosis of growths in the large intestine is full of interest and surprises which may be unpleasant and dangerous.

Beginning at the cæcum, either a malignant or tuberculous tumour, by interfering with the ileo-cæcal opening, may cause "attacks of pain," and the discovery of a lump, in the majority of cases, suggests appendicitis. It requires careful consideration of the history and of the physical signs if this error is to be avoided, and with both of these it is possible to make a mistake. More than once I have opened an abscess in the right iliac fossa expecting to remove the appendix and have found a perforating malignant ulcer of the cæcum, and more than once, expecting to remove the cæcum for cancer, I have found appendicitis and an old abscess.

In women a large ileo-cæcal tumour due to cancer or tuberculosis is usually thought to be an ovarian tumour till the abdomen is opened, and if the operator should chance to be a specialist in gynæcology the predicament is awkward for his patient and for himself. I have already mentioned and quoted a case in support of the statement that an annular stricture of the ascending colon immediately above the cæcum causes a sausage-shaped tumour, with an upper end, the stricture, and a lower, the end of the cæcum, which hardens when distended (the ileo-cæcal valve being competent) and relaxes when empty, accurately simulating the tumour of an intussusception. Higher in the ascending colon a malignant growth resembles very closely a tumour of the kidney.

At the hepatic flexure a growth resembles closely one of the gall-bladder.

In the transverse colon it is not seldom taken to be cancer of the stomach, and I have seen this error made by a most distinguished surgeon.

In the descending colon, again, the kidney or the spleen may be suspected, and in the sigmoid flexure there is another trap for the gynæcologists, who, finding a hard painful lump in Douglas's pouch, mistake it for a diseased and prolapsed ovary.

Two of the recent additions to diagnosis of growth in the large intestine and stricture are the sigmoidoscope for those just out of reach of the finger, and bismuth injections with X-ray pictures to show stricture anywhere in the colon. The value of the sigmoidoscope is universally conceded, that of bismuth injections is not so well known. Recently at the clinic of Professor Tuffier in Paris I saw X-ray pictures showing stricture of all parts of the colon so clearly that no mistake was possible.

Treatment of Intestinal Obstruction.—It is as true to-day as it was ten years ago when in the article I wrote for the *Encyclopædia Medica* the opening sentence on operative treatment, "In cases of acute intestinal obstruction the sooner operation can be performed the better the prognosis," and it is unlikely that this will ever prove other than the best guide.

Since then I have emphasised this view by figures which support it so strongly as to bear repetition. Amongst other evidence [1] the following occurs:—"During the past 3 years, from 1900 up to date, the registrar has found a note of 11 patients who were attacked (in hospital) by acute intestinal obstruction whilst under my care, and suffering from other conditions. Of these 9 recovered and 2 died. One death occurred in a patient who was not operated upon from mistaken diagnosis. Of 10 patients operated upon 9 recovered and 1 died. The patient who died was a young man whose appendix had been removed two days previously, and who developed a complicated volvulus involving the small intestine."

That early operation is only done now when a definite diagnosis can be made by physician and practitioners is certain, and it is the surgeons chiefly who must teach how this is to be done, as well as its importance. If anyone doubts the truth of this let him consider the results, and as compared with those of twenty years ago in cases of intussusception. Early diagnosis and prompt operation

[1] See an address on the Treatment of Abdominal Emergencies, *Lancet*, 31st January 1905.

have converted this deadly complaint into one almost devoid of risk.

So soon as physicians teach that every patient with more than ordinary abdominal pain must at once be removed to a properly equipped hospital for observation and treatment, the mortality from abdominal emergencies will drop more than 50 per cent. Every surgeon knows that these emergency operations are usually easy, that the operations from the surgical point of view are safe, and that their mortality, still dreadful, is the result of delay and not of surgical difficulties.

It is not *always* possible to make a correct differential diagnosis in these cases, and, humiliating as the position is, it is of the first importance that it should be accepted and acted upon.

My own practice for years in doubtful cases has been, first, to have the stomach washed out by tube. I have converted thus a mere suspicion into a certainty by finding feculent stomach contents in the stomach of a patient who had never vomited. The next is to administer a large dose of castor oil and later an enema. The effect of this in intestinal obstruction is to stir the intestine up to activity and often to make the diagnosis definite. Then operation is undertaken at once. It is not always possible to say that a case is certainly one of intestinal obstruction, still less to say where the block is and of what nature; but if a complete diagnosis can be made the surgical treatment becomes safer and easier because the abdomen can be opened directly over the site of the lesion. With regard to the operation I can add little to what I wrote in the *Encyclopædia of Medicine and Surgery* (vol. iv.), but would like to reiterate as to the importance of a carefully carried out scheme in exploratory operations, and to emphasise the danger of allowing the intestines to escape from the abdominal cavity in these obstruction cases. If the assistant is given to understand that his chief duty, and one he must not neglect for any other, is to keep the intestines confined within the abdomen, the mortality following operations for acute obstruction will be considerably diminished. Another point concerns the treatment of distended intestines. If these can be kept within the abdomen no fears need be entertained for them; if they escape it is necessary to provide for their artificial relief from distension. I do not trust multiple incisions or rapid emptying through a tube, but prefer to leave a tube in and do an enterostomy. If a little care is taken with this the fæcal fistula resulting ought to close spontaneously. The care

required is to place a purse-string suture round the intestinal opening, and after passing a tube into the intestine, to tighten up the suture and *to tuck in the intestinal coats round the tube* so that no mucous membrane projects and a fistula lined by peritoneum is left when the tube is taken away. It is fortunately becoming rare now to see cases left so long as was the custom only ten years ago, but for a patient very ill with a tense distended abdomen the best chance I still believe is given by a simple enterostomy. The greatest addition to the radical surgery of obstruction in the small intestine has been made by Professor A. E. Barker, who advocates excision of a considerable length of distended bowel above the obstruction.

Where the obstruction, even though acute, is due to block of the large intestine, complete exploration may be made with impunity. In these cases all surgeons are now agreed that no attempt should be made to do a radical operation till after obstruction is relieved. A radical cure then means three operations—one for drainage, the second to remove the growth, and the third to close the drainage opening, unless this is made in such a position as to be removable with the growth, which, I think, is undesirable, or is made valvular so that it may close spontaneously. The exploration allows of a choice of sites for the colostomy, and in inoperable cases this will be made as low down as possible. Where it is decided that a radical cure should be attempted I prefer cæcostomy, because it is most easily done, is away from the future site of operation, and the opening can be readily and safely closed when no longer needed.

With regard to the "radical" operation, I would like to say that in my view the ideal one is suggested by the excellent work of Jameson and Dobson on the colon lymphatics, and I feel no doubt that this is the standard we should keep before us. There has been a larger mortality in my cases than I could have wished, but all new operative methods pass almost insensibly from this stage into others of greater and greater success as experience of them increases, and none of us can say exactly why or what has made the difference. Though a high standard should be set, it must not be allowed to dominate the judgment in individual instances. Experience has proved that in a considerable percentage of cases permanent cure has followed much more limited excisions than are suggested and this knowledge must be used for the benefit of patients judged to be unlikely to do well after the more formid-

able undertakings. At the same time it should be remembered that mobilisation of the colon by division of the outer and upper peritoneal layer of its mesentery has made dangerous operations safe and difficult ones easy. In addition to the possibilities of doing all excision and suture work outside of the abdomen, it has made the blood-supply visible and made lateral anastomosis, which I still think the best form of junction, suitable to any part of the colon. In mentioning anastomosis I have to confess that I think it may *sometimes* be made more satisfactorily by an apparatus than by simple suture, and I am not above availing myself of the help of the most ingenious of such devices, the Murphy button. Instead of this, when time has been an important factor, I have used a simple glass bobbin (Fig. 1), and in the notes of one of my two reported cases I find that with this the anastomosis was made in 5 minutes, and that both patients recovered. The other apparatus to which I wish to draw your attention (Fig. 2) was described in a paper read before the Gynæcological Society of London, and published in the *British Gynæcological Journal* of February 1901. It was designed to deal with those difficult cases of growth in the pelvic colon too low down to be dealt with in the ordinary way from above and too high to be accessible from below. It was used in 4 of my cases last year, with 3 recoveries and 1 death, and should, I think, be better known. In one of my cases a stricture followed its use, but this appeared to result from prolonged sepsis. I know of nothing else against this method.

In operable cases an anastomosis has so many advantages over permanent colostomy that it is a duty to perform it when possible. Within the last year I visited the operating theatre of a colleague and found him engaged in joining the ileum to the transverse colon in a case of inoperable cancerous stricture of the hepatic flexure. He did not know, or could not believe, that if his operation was successful and the ileo-cæcal valve competent the cæcum would burst, though I have taught this for at least 15 years. If I have performed this anastomosis, at the same time I have done a "pyloroplasty" on the valve.

In connection with these desultory remarks I am giving the statistics of all my operations for intestinal obstruction, active or potential, for 1910, and abstract notes of every case. They illustrate every point I have endeavoured to make, and fill up many gaps. The notes have been supplied by the Surgical Registrar, Mr Willan, and edited by Mr Saint, to whom I am under great

obligations. It may add to their interest that with each case the pre-operative and post-operative diagnosis have been given. For many years it has been my rule that *one* definite diagnosis should be made and written down before operation, and I have found this a very healthy stimulus, for in the great majority it is my own opinion that is recorded. It may also interest many to find that surgeons are not *always* right, and to suggest after careful perusal of the notes how they may have gone wrong.

Before concluding I would like to quote the final words of a clinical lecture of mine given in the Royal Victoria Infirmary on 10th June 1909. "The results in cases of acute obstruction of the small intestine are little, if any, better than they were twenty years ago; whereas the results in chronic obstruction, especially affecting the large intestine, have enormously improved. Why is this? It is because operative *technique* has made such strides during that time, and operative skill has a considerable influence on the statistics of colon obstruction. Operations for colon obstruction mostly require the difficult operation of resection for their radical cure, and this has been perfected during that time. But operations for the radical cure of small intestine obstruction have not been influenced much, if at all, by surgical progress. They are nearly all simple—*e.g.* the division of a band, the separation of adhesions, the withdrawal of a strangulated loop, and these could be done as successfully twenty years ago as they can be now.

The lesson we learn is, that early diagnosis and immediate operation are of far greater importance than operative skill, and no improvement in results can be expected until this has been acknowledged and acted upon. Then patients will recover from acute intestinal obstruction as they now recover after operations for strangulated hernia."

STATISTICS

Operations for Cases of Intestinal Obstruction during 1910

ACUTE :

I. No Mechanical Agency.	1. Paralytic ileus—albumen in the urine. 2. Paralytic ileus in a case of phthisis—nothing in abdomen.
II. Kink—No Strangulation.	3. Adhesion from old appendix—freeing and cure of hernia. Second operation, acute kink—death. 4. Appendix on left side of spine—acute kink—44 ins. resection. 5. Adhesions from old appendix—kink not acute—intestinal obstruction. 6. Interval appendix—kink from numerous adhesions, not acute.
III. Strangulation.	7. Strangulation under a band—symptoms less than expected. 8. By band, in association with old appendix abscess. 9. Adhesions in pelvis, with volvulus (secondary) after puerperal sepsis. 10. Strangulation of loop by Meckel; Meckel gangrenous. 11. Adhesions after strangulated femoral hernia—fistula present. 12. Fistula in strangulated ventral hernia after appendix; 9 ft. resection.
IV. Large Bowel.	13. Inoperable carcinoma of ileo-cæcal valve. 14. Carcinoma of ascending colon, immediately above cæcum, with ovarian cyst.

CHRONIC :

I. Adenomatous Polypus.	15. Small intestine, with chronic intussusception. 16. Sigmoid—case previously treated for mucous colitis.
II. Ileo-cæcal Tuberculosis.	17. Diagnosed chronic appendix abscess—1. Excision of mass and enterostomy. 2. Acute obstruction relieved. 3. Lateral anastomosis and closure. 18. Old operation for appendix ?—fistula—Jameson-Dobson. 19. Tubercle and chronic appendix—excision, not Jameson-Dobson. 20. Tubercle ?—1. Lateral anastomosis. 2. Jameson-Dobson.

III. Carcinoma.

21. Caput cæci—intussuscepted into ascending colon—Jameson-Dobson.
22. Ascending colon—about middle—Jameson-Dobson—death from dilation of stomach.
23. Ascending colon—practically no bowel symptoms—diagnosed kidney.
24. Hepatic flexure—spreading from gall-bladder by adhesion.
25. Transverse colon—excision and lateral anastomosis.
26. Splenic flexure—inoperable, permanent colostomy.
27. Sigmoid flexure—1. Cæcostomy. 2. Excision and left colostomy. 3. Closure of cæcostomy.
28. Sigmoid flexure—excision and permanent colostomy.
29. Sigmoid flexure—1. Sigmoidostomy. 2. Excision and telescoping ends.
30. Sigmoid flexure—typical history—excision and end-to-end telescoping.
31. Sigmoid flexure—diagnosed "primary ovarian." Pathologist "primary bowel."
32. Sigmoid flexure—complicated by vesico-colic fistula.

POTENTIAL:

33. Multiple polypi of rectum—colostomy.
34. Multiple polypi of colon, from middle of transverse to the rectum, associated with ulceration—cæcostomy—death.
35. Sigmoido-vesical fistula—sacculitis and perforation.
36. Gastro-colic fistula—carcinoma of stomach, inoperable.
37. Recto-vaginal fistula—secondary peritoneal deposits, reported cancer.

DIFFERENTIAL DIAGNOSIS:

38. Perforated duodenal ulcer, three days' duration, with general peritonitis; admitted with retention of urine and paralytic ileus. Diagnosed obstruction.
39. Old appendix abscess—diagnosed ileo-cæcal carcinoma.

Of the total number of cases operated on for intestinal obstruction, viz., 39, there were 10 deaths, *i.e.* 25.7 per cent.

Of cases in which resection of gut was performed—20 in number—4 died, *i.e.* 20 per cent. A detailed list of these is given in tabular form.

CASES IN WHICH RESECTION OF GUT WAS PERFORMED

Bowel.	Case No.	Sex.	Age.	Disease.	Operation.	Recovery or Death.	Cause of Death—if died.
SMALL	4	M	33	Kinking from appendix	Excision of 44 ins.	R	...
	7	F	12	Strangulation under band	,, 2 ft.	R	...
	10	M	24	Strangulation due to Meckel	,, 3 ft.	R	...
	12	F	50	Fistula with strangulated ventral hernia	,, 9 ft.	R	...
LARGE—*Acute*	14	F	62	Carcinoma of ascending colon	Jameson-Dobson	D	Shock — enterostomy did not act
Chronic	16	M	58	Malignant papilloma of sigmoid	Excision of bowel involved	R	...
	17	M	30	Ileo-cæcal tuberculosis	Excision, &c. — 3 operations	R	...
	18	F	20	Ileo-cæcal tuberculosis	Jameson-Dobson	R	...
	19	M	48	Ileo-cæcal tuberculosis	Excision	R	...
	20	M	18	Ileo-cæcal tuberculosis	Jameson-Dobson — 2 operations	R	...
	21	F	64	Carcinoma of caput cæci	Jameson-Dobson	R	...
	22	M	43	Carcinoma of ascending colon	,,	D	Acute dilatation of stomach
	23	M	59	Carcinoma of ascending colon	,,	D	Anastomosis given way; acute dilatation of stomach
	25	F	44	Carcinoma of transverse colon	Excision and lateral anastomosis	R	...
	27	M	62	Carcinoma of sigmoid colon	Excision and colostomy—3 operations	R	...
	28	F	64	Carcinoma of sigmoid colon	Excision and colostomy—1 operation	R	...
	29	F	75	Carcinoma of sigmoid colon	Excision — 2 operations	D	Mania, 18 days later
	30	F	42	Carcinoma of sigmoid colon	Excision—1 operation	R	...
	31	F	45	Carcinoma of sigmoid colon	Resection of 4 ins. of adherent infiltrated small gut	R	...
	35	F	61	Sigmoido-vesical fistula	Resection of adherent sigmoid and ileum—9 ins.	R	...

CASE .—*Paralytic Ileus.*—A man, aged 26, was admitted to the medical side, Royal Victoria Infirmary, for the treatment of pulmonary phthisis, 1 6 10.

On 20 8 10 I was asked to operate upon him for intestinal obstruction of a few days' duration. On opening the abdomen nothing was found to account for his symptoms. He recovered

promptly from the operation, had no further abdominal trouble, and was transferred back to the medical house.

Case .—*Congenital Inguinal Hernia—Secondary Volvulus due to Adhesions from Appendicitis—Operation—Recovery. Second Operation for Intestinal Obstruction—Due to Adhesions—Death.*—J. C., aged 30. Admitted 18/2/10. Discharged 7/3/10. Re-admitted 30/4/10. Died same day.

Patient had had a rupture as long as he could remember. He had never had any difficulty with the reduction of it. He had occasionally had some discomfort when it increased in size, but no vomiting or stoppage in the bowels, and was always relieved by reduction of the mass. Eighteen months ago the patient was seized with intermittent abdominal pain, gradually increasing in severity, accompanied by vomiting, constipation, and inability to pass flatus, while at the same time he noticed lumps rising in the belly with the pains and disappearing again ; there was slight rumbling with the onset, but it was not present later. He described his feelings as though there were some wind which wanted to break away. The pain passed off quite as quickly as it came on, and with the first attacks he felt all right after they had subsided. The first attacks were relieved by enemata, but not the last. He had had in all 5 such attacks, coming on at varying times of the day, the last, a week ago, the worst he had had. It was unrelieved by enemata and made worse by aperient medicine ; it lasted for 8 hours. Usually his stools consisted for the most part of small hard fæcal masses, and he said he had noticed flattening of the motion. He had had no alternating diarrhœa and constipation, and only once, 1½ hours after onset of attack, he had some increase in the frequency of his stools ; with these attacks he had had no pain in his rupture, and had had no irreducibility of it, the swelling being easily returned to the abdomen.

On admission the patient was in fairly good general condition ; temperature and pulse normal ; chest clear.

Local.—He had a right complete reducible inguinal hernia-congenital sac ; nothing abnormal felt in his abdomen. Per rectum, nothing abnormal palpated.

Diagnosis.—Right reducible congenital inguinal hernia, with a volvulus of the lower ileum, probably due to adhesions resulting from a previous attack of strangulation in the hernia, the adhesions thus being in the right iliac fossa.

Operation—22 2 10. *Mr Morison*—Oblique incision in lower part of right iliac region, parallel to Poupart ; abdomen opened. Adhesion found extending from the appendix to a piece of the ileum at its lower part. A tuberculous gland was situated in the mesentery of the ileum just beneath the attachment of the adhesion from the appendix. There was a well-marked scar on the wall of the ileum, resulting from a twist of the gut from left to right, evidently due to the adhesion. The mesentery also of that part of the ileum, which had been twisted, was hard and fibrous, like an ovarian pedicle that had undergone torsion.

The adhesion was separated and the tuberculous gland, the size of a small nut, was removed ; the appendix was also removed and the stump buried—it was internal and posterior to the cæcum, pointing towards the pelvis. The original skin incision was extended and a radical cure of the hernia performed in a modified Halsted-Kocher method. The sac was thin and contained no gut, but the testicle, only partially descended, was lying at the bottom of it. Belly wall closed in layers.

Microscopic Report of Gland.—Small lymph gland infiltrated with lime salts and showing diffuse fibrosis ; some caseation is present, but no giant cells, though the condition is probably old tuberculosis.

7 3 10.—Uninterrupted recovery. Left hospital all healed.

Readmitted 30 4 10 with intestinal obstruction of 3 days' duration ; he was very ill on arrival, with belly tense and distended, and pulse 140.

Operation—30 4 10. *Mr Morison.*—Median infra-umbilical incision ; abdomen opened. Numerous adhesions found in ileo-cæcal region. Those which appeared to be giving rise, by kinking, to the obstruction were separated and the bowel freed. Belly closed.

On return to the ward patient collapsed and died in the afternoon.

Post Mortem.—Nothing found, except other adhesions in ileo-cæcal region, fairly widespread. Death from exhaustion due to delayed operation.

Case .—*Erratic Appendix—Appendix Abscess on Left Side causing Intestinal Obstruction—Resection of* 44 *ins. of Damaged Intestine—Recovery.*—J. K., aged 33, overman. Admitted 7 1 10. Discharged 11 2 10.

Three days ago patient developed a pain in his abdomen, more marked on his left side ; it came on about 2 P.M. while he was outside. He thought it was wind and stayed out till evening.

It was intermittent, but increasing in severity, and as it became worse it became practically continuous. At night he took some aperient and an hour or two later vomited for the first time. The aperient was repeated the following morning and vomiting again occurred. Since this he had vomited frequently and the later vomitus had been fæcal. There had been some, but not marked, rumbling in his abdomen, and he had belched up a great deal of wind. He had not had his bowels moved of themselves and at first there was complete arrest of flatus. He had had 3 enemata, each 3 pints, giving rise to very little result, though the last two were returned accompanied by a great deal of flatus, which relieved the distension that had occurred. Since this he had been much easier, that is, for the last 24 hours.

On admission patient looked somewhat worn out, features drawn and sunken; tongue, dirty brown fur; temperature, 98°; pulse, 98; chest, clear. Vomited a little after admission, vomited matter somewhat foul-smelling.

Local.—Abdomen distended, but quite lax; some tenderness to the left of the mid-line, but no rigidity; distension was in front and not in the flanks; no free fluid made out; peristalsis, very little, at times a slight change in the contour of the abdomen; no hernia present; urine contained albumen $\frac{1}{2}$. For this reason operation was negatived, the suggestion offered by the surgeon called in being that of paralytic ileus due to kidney disease. Mist. purg. alb. ʒi. every hour was ordered; bowels were moved twice before morning. 9.15 A.M., patient still feeling sick, but no more vomiting. Patient was transferred to the medical wards.

28/1/10.—Seen by Mr Morison; patient had been having considerable pain in the abdomen, intermittent and cramping, associated with sickness, suggestive of some partial obstruction.

Local.—A mass was felt on deep palpation to the left of the umbilicus, about the region of the duodeno-jejunal junction; tender on palpation indefinite in shape.

Diagnosis offered.—Tuberculous glands in the mesentery causing adhesions and intestinal obstruction. Urine now contained no albumen and none had been present for some time.

Operation—29/1/10. *Mr Morison.*—Abdomen opened through the left rectus sheath, the muscle being displaced outwards. On exploring the small intestine with the finger a portion was arrived at which was adherent in two distinct puckered places to the mesentery on the left of the spine—these coils were reddened,

œdematous, and covered with yellow lymph. On separating them an old abscess cavity without pus was exposed between the intestine and posterior abdominal wall. At the bottom of this cavity was seen what looked like an enlarged gland, but which, on pulling up and separating, proved to be the bulbous tip of the appendix. After fully separating it, it was found to run behind the lower ileum to its cæcal attachment; it was removed in the usual way and the stump inverted by purse-string suture. The mesentery of the released gut and the gut itself were deemed too much damaged to leave, so 44 ins. were resected. The bowel was divided by the thermo-cautery between clamps; the two ends were closed by through-and-through catgut and overlying lemberts of silk. A lateral anastomosis was now performed by simple suture. A mass of enlarged glands was palpated in the mesentery, but these were not interfered with. Belly wall closed in layers. Patient was transfused to the extent of 35 ozs. of saline during the operation.

Specimen.—Appendix was thickened and œdematous, especially the mucous membrane, which was very thick and grayish-looking. No perforation detected. Mr Morison thought it might be tuberculous and that the enlarged mesenteric glands were either of this nature or septic. On examination of the intestine resected a perforation was discovered—not the result of accident during the separation. This Mr Morison considered to be the site at which the abscess, previously existing, had discharged itself. If the gut had not been resected this opening would have been missed and leakage would have followed the operation.

Microscopic Report of Appendix—3 1739.—Chronic inflammatory thickening of submucous and peritoneal coats—no evidence of tuberculosis.

11/2 10.—Patient made an absolutely straightforward recovery. He is now well; bowels acting all right and he is eating solid food; wound healed; left hospital. Three months later returned for inspection; wound excellent; has put on about 1½ stones in weight; bowels have acted every day since his discharge.

Case .—*Intestinal Obstruction due to Kinking from Adhesions following Appendicectomy.*—H. J., aged 43, cartman. Admitted 2/2/10. Discharged 21/2/10.

Patient was admitted complaining of severe abdominal pain.

Six and a half years previously he had his appendix removed; at the operation the omentum looked as though it were becoming

gangrenous, but it was not interfered with ; there was no abscess. The appendix was acutely inflamed but not perforated.

For the first 3 years he was quite well, but subsequently he was troubled with attacks of pain at intervals. The day before his admission, while at work, he was seized with sudden severe pain in the region of the umbilicus. It passed off towards night, but returned with increased severity next morning, at about 7 A.M., and after this became worse. He had no vomiting with it, and his bowels had been quite regular, being moved in the morning before the onset. He was able to pass flatus. There had been no pain with micturition.

On admission he was in very good general condition ; temperature, 99° ; pulse, 61.

Local.—He was tender in the umbilical region, and there was some resistance to examination, indefinite in character. He was quite resonant all over, and no tumour was felt. No peristalsis was visible.

Diagnosis.—Intestinal Obstruction ?

Operation—12/2/10. *Mr Morison.*—Median laparotomy performed, excising the umbilicus. On opening the abdomen and exploring the ileo-cæcal region the last part of the ileum was found adherent by its antimesenteric border to its own mesentery, thus producing a distinct kink. The adhesion was separated. The old appendix site was examined and found to be an excellent result. Abdominal wound closed in layers.

21/2/10.—Patient made an uninterrupted recovery and left hospital, the wound having healed by first intention.

Case .—*Intestinal Obstruction from Adhesions and Kinking following Appendicostomy.*—A single girl, aged 25, was admitted to the Royal Infirmary, 9/7/10.

History.—Fifteen months previously her appendix and right ovary had been removed at home for what she had been told was appendicitis. For six months after the operation she was quite well, when she began to have pain in her right side, accompanied by rumblings loud enough for those in the room to hear, and relieved by the passage of flatus. Her bowels were constipated and her appetite poor, but she was not getting any thinner.

On admission the patient was a spare woman in fair health.

The abdomen showed an old scar over the linea semilunaris

and below the umbilicus. At its upper end there was some rigidity of the muscles, with a tender resistant area underlying it.

Diagnosis.—Ileo-cæcal tuberculosis.

Operation—13/7/10.—The abdomen was opened by an oblique incision in the right iliac fossa. Fluid escaped. There were numerous firm adhesions around the cæcum and lower ileum which were divided by scissors between clips. After freeing the bowel the abdomen was closed. Recovery.

Case .—*Strangulation of Intestine under a Tight Band due to Tubercle—Resection of 2 ft. of Small Intestine.*—A school-girl, aged 12, was admitted to the Royal Victoria Infirmary, 4/11/10.

History.—She was quite well till 3 days ago (1st November). On that day, on her return from school, she was seized with a pain in her stomach which made her feel sick and vomit. The pain soon became intermittent in character, and continued till her admission. The pain had never been sufficient to make her shout or to keep her awake. There had been rumbling of wind in the bowels, but no flatus had passed since the attack commenced. Any food produced pain and vomiting at once, and the sickness entirely relieved the pain. The vomited matter was only the food she had taken ; there was no bile nor blood in it. Her bowels were slightly moved after an enema the night before admission. There was no blood in the motion.

On admission she looked somewhat pinched, but had a good colour, and did not appear to be seriously ill. Temperature, 99.4° ; pulse, 126.

The abdomen was distended and tympanitic except in the flanks, where there was shifting dulness. An indefinite tender mass could be felt in the right iliac and flank region.

Nothing was discovered per rectum, and the examining finger was not blood-stained.

Diagnosis.—Subacute intussusception.

Operation.—The abdomen was opened in the mid-line by excising the umbilicus. Some clear serous fluid escaped, and a deeply congested loop of strangulated intestine presented. The tumour was discovered in the right flank, and was turned out on to the abdominal wall. It was then seen to be composed of a bunch of strangulated gangrenous intestine which had passed through a cord-like ring of adhesion not bigger than a wedding ring. Two feet of small intestine immediately above the ileo-cæcal

valve, half of it strangulated as described, were excised. In making the separation a tuberculous gland to which the adhesion was attached was accidentally opened. Its contents were scooped out and the capsule was seared with a thermo-cautery. The divided intestinal ends were then closed, a lateral anastomosis by two layers of sutures made between the ileum and cæcum and the abdomen was closed in three layers without drainage. Recovery.

Case .—*Intestinal Obstruction from Band due to old Appendix Abscess.*—A school-boy, aged 13, admitted to Royal Victoria Infirmary, 30/5/10.

History.—For 3 years the patient had been subject to recurrent attacks of abdominal pain. It came on quite suddenly, was of a stabbing character, and diffused over the whole abdomen. Vomiting of yellow fluid, which did not relieve the pain, was always present, and he could neither pass fæces nor flatus The attack was always removed by the passage of flatus.

On admission he was an emaciated, weedy-looking boy. Pulse, 96 ; temperature, 100° F.

Abdomen.—Nothing could be felt from above. The rectum was balooned. Anteriorly a hard mass could be felt between it and the base of the bladder like an inflammatory exudate.

Diagnosis.—Intestinal obstruction due to tuberculous adhesions.

Operation—31 5 10.—The abdomen was opened in the middle line after excision of the umbilicus. An adherent strangulated coil of obstructed small intestine was found in the right iliac fossa with a band surrounding it. The intestine above was much enlarged and hypertrophied, below it was small and contracted. It was freed from the adhesions, exposing the remains of an abscess which was traced to the vermiform appendix. The raw surface of the intestine was sutured, and the appendix, which was adherent in the pelvis, was excised. There were several tuberculous glands in the mesentery of the small intestine, but these had nothing to do with the present condition. The abdomen was closed in layers. The appendix contained pus, and had a stricture at the cæcal end. Recovered.

Case .—*Subacute Intestinal Obstruction from Adhesions following Puerperal Sepsis—Acute Termination due to Secondary Volvulus.*—M. J. B., aged 28. Admitted 27/1/10. Died 3/2/10.

Pain.—Four days before admission the patient was awaked

at 5 A.M. with cramping pain in the abdomen, which gradually increased in severity; it was intermittent in character, and relieved by pressure. She vomited repeatedly everything she took, and also between the periods of taking food, at first bile, and later dark-brown fœtid material—this had continued. The vomiting relieved the pain for a short time. Rumbling was present, quite loud and audible, whenever the pain came on severely.

Arrest of Flatus.—Since the onset she had passed no flatus, but she had belched up a great deal of wind. Her bowels had not been moved since the beginning of the attack. Symptoms continued up to the day of admission, but she had been somewhat easier since 9 A.M.

Previous.—There years ago patient had a 7-months child, and she was in bed ill for a fortnight with abdominal pain.

On admission patient looked ill, with pale and drawn countenance; temperature normal; pulse very soft and weak; tongue thickly furred; chest clear.

Local.—Abdomen distended; oblique ladder pattern of peristalsis marked, and numerous rumblings heard. Per vaginam, uterus felt retroverted and fixed behind to the rectum.

Gastric lavage was performed. A great quantity of very foul fæcal matter was drawn off, which gave her immediate relief.

Diagnosis.—Intestinal obstruction in small intestine, due to kinking of the gut, from adhesions in the pelvis, as a result of old pelvic peritonitis. Too ill for a radical and not ill enough for a palliative operation. Palliative treatment was adopted by lavage, poultices to the abdomen, and nutrient enemata. She passed some flatus, and had a free evacuation; she was not wholly relieved, though much better. On the early morning of operation, 1 2 10, all her symptoms returned acutely. *Mr Morison.*—Small incision in right iliac fossa; piece of distended small intestine drawn out and fixed. Paul's tube tied in. 3 2 10.—No fæces have passed through the enterostomy, despite the introduction of a large quantity of saline into the bowel. Death.

Necropsy.—On opening abdomen the small intestines were hugely distended. On tracing the dilated gut down the obstruction was arrived at on the right side of the pelvic wall. Here a strong band of adhesion bound a loop of the lower ileum to the lateral pelvic wall, and about 3 ft. of gut, black and nearly gangrenous, was found twisted on itself, lying in the pelvis (secondary volvulus); numerous other small adhesions were present, con-

necting the small bowel with the fundus uteri; the uterus was retroverted and bound to Douglas's pouch and the rectum by adhesions. On removing the twisted bowel about 5 ins. of the lower ileum and the cæcum were normal, but collapsed, and above this was the twisted portion, about 2 ft. in length, the distended gut being higher still. The enterostomy opening was in a distended portion of the gut, not far from the obstruction. It had not emptied the paretic bowel.

Case .—*Strangulation due to a Meckel's Diverticulum—Meckel Gangrenous.*—G. T. H., aged 24, a bricklayer, was admitted to the Royal Victoria Infirmary on 20th October. Discharged 7 11 10.

History of Present Illness.—It commenced suddenly 34 hours before admission, 1½ hours after his tea. He was seized with a very severe cramp-like pain across his bowels, bad enough to make him shout out and sweat. Vomiting commenced 2 hours afterwards, and continued during the whole night. The vomiting did not relieve the pain. He had a feeling that if he could only pass wind downwards it would relieve him. The pain and vomiting and sweating continued till his admission.

Enemata were admimistered soon after his illness commenced, but no relief or discharge of fæces or flatus had resulted. He had no urinary trouble.

Previous Health.—Had been good. He had never suffered from " indigestion " at all.

Condition on Admission.—The patient had the appearance of a strong healthy man acutely ill and suffering greatly. He was lying on his back with both his legs drawn up.

His tongue was moist and furred; pulse, normal; temperature, 96° F.

Abdomen.—On inspection the whole abdomen was distended, and respiratory movements were absent. There was no localised swelling or visible peristalsis. On palpation it was tender all over, and there was marked rigidity of the whole muscular covering. (Four hours previously it had been noted that rigidity and tenderness were limited to the right side.)

On percussion both flanks and a semilunar area over the pubis were dull, and the dulness changed with every change of posture. (Four hours previously the dulness had been limited to the right side, the left being hyper-resonant.)

Per Rectum.—There was fulness in front as of fluid in the recto-vesical pouch.

Diagnosis.—All agreed that the patient had acute general peritonitis, with a considerable quantity of free fluid in the abdomen. The cause of this was not so clear, though the physical signs and the sex and age of the patient suggested ruptured duodenal ulcer. The only things against this were the absence of any previous history of indigestion and the constant vomiting since the attack. It seemed difficult to explain the early general peritonitis, the presence of so much fluid, and the absence of more definite evidence of increased peristalsis if the case was one of acute intestinal obstruction, as had been suggested.

Operation. Professor Rutherford Morison.—The abdomen was opened in the middle line by excision of the umbilicus. A large quantity (about a quart) of very foul-smelling greyish-coloured fluid escaped. The stomach and duodenum were normal. The incision was prolonged downwards, great care being taken to keep all the intestines in the abdomen ; a mass of distended acutely strangulated bowel with what was clearly a distended gangrenous Meckel's diverticulum was exposed on the right side. The distended sodden bowel for about 3 ft. above the obstruction was withdrawn from the abdomen along with the portion strangulated, and the whole was excised.

The lower or distal end of the bowel was divided about $\frac{3}{4}$ of an inch above the ileo-cæcal valve, and both proximal and distal ends were tightly tied together with silk over a glass bobbin. This was pushed on (intussuscepted) into the cæcum, and fixed there by Lembert's sutures, attaching the ileum to the cæcum. (The anastomosis by the bobbin required 5 minutes.)

The abdomen was washed out with abundance of hot normal saline (℥i. of sodium chloride to 1 pint of water), a quantity of dirty fluid escaping from the pelvis, and it was then closed entirely by tier suture.

All of the visible intestine and the parietal peritoneum were bright red, but neither had lost its gloss, and there were no lymph deposits.

As soon as the operation started intravenous transfusion was commenced, and continued slowly during the whole procedure.

Pathology.—A band was found passing from the apex of the Meckel, to be attached to the mesentery close to the base of the diverticulum. The Meckel was twisted on itself, producing gan-

grene, and a loop of bowel had become strangulated under the band.

After-Progress.—On the day following operation the patient had the appearance of a man entirely relieved of a serious illness, and except for some suppuration of the abdominal wound made a straightforward recovery, and went home on 7th November.

Case .—*Strangulated Gangrenous Hernia—Recovery—Intestinal Obstruction due to Adhesions resulting in Gangrene of Gut—Resection—Recovery.*—J. D., age 39, single. Admitted 18/4/10. Discharged 29/4/10. Readmitted 30/5/10. Discharged 29/6/10.

Patient was admitted complaining of a swelling in the right groin, associated with pain and vomiting.

For 7½ years she had had a rupture at that site, which appeared spontaneously and gradually increased in size. For several years she had had slight attacks of pain in it, and had a very severe one 7 weeks before admission. From that time the pain had persisted more or less at intervals. About 2 P.M. on the day before admission she was seized with a very severe attack of pain, at first localised to the hernia, and later referred to the umbilical region and lower abdomen. Vomiting accompanied the pain, and persisted. The vomitus at first consisted of the stomach contents, but on admission had become bilious. She had her bowels moved during the morning before the onset of the attack, but subsequently passed neither fæces nor flatus.

On admission her general condition was poor, her face was pale and drawn, tongue covered with a yellow fur. Temperature, 97°; pulse, 64.

Local.—There was a swelling the size of a large egg situated below the right Poupart and external to the pubic spine, over the site of the femoral canal. It was tense, hard, extremely tender, smooth on the surface, irreducible, and without impulse on coughing.

Diagnosis.—Strangulated femoral hernia.

Operation—18/4/10. *Mr Morison.*—Oblique incision below Poupart, sac of hernia separated and opened. On exposing the gut it was almost black, but on freeing the constriction, by dividing Gimbernat's ligament, it recovered its colour somewhat. It was returned to the abdomen just within the femoral ring with a large tube in contact with it. The sac, which was œdematous, was packed with gauze, and no attempt was made at a radical cure,

the wound being allowed to granulate. A good pad with firm pressure was applied.

29/4/10.—Wound almost healed; patient went home.

After leaving hospital she was quite well for a week; she then had a sudden attack of acute pain in the abdomen and vomited some mucoid fluid. With the attack her abdomen swelled up and there were loud rumblings. Her bowels were constipated and the passage of flatus gave her temporary ease. Subsequently she had a similar attack almost every evening.

On readmission she looked in fair general condition; tongue was moist and furred; temperature, 98.6°; pulse, 88.

Local.—There was a small sinus present in the middle of the scar in the right groin. On examining the abdomen definite peristalsis of the oblique ladder pattern was visible, which, on palpation, appeared to culminate in the region of the right iliac fossa. There were numerous rumblings in addition, also in the right iliac fossa. Associated with this she had repeated vomiting.

Diagnosis.—Intestinal obstruction due to adhesions in connection with the old hernia.

Operation—7/6/10. *Mr Morison.*—An incision was made, extending from the anterior superior iliac spine to 1 inch from the symphysis pubis in the right iliac fossa. On opening the abdomen the ileum, about 3 ft. from the ileo-cæcal valve, was found adherent to the femoral canal; above, the intestine was distended, injected, and hypertrophied; below, it was thin and pale. On separating the adherent gut it was found that only the mesenteric side of the intestine was left, the convexity and sides of the gut had disappeared (gangrene and absorption). The bowel above and below this region was clamped and divided by the thermo-cautery. End-to-end anastomosis was performed by a Murphy's button, after resection of the damaged intestine. Abdominal wound closed in layers.

21/6/10.—Patient made a slow but good recovery, and left hospital healed and well.

Case .—*Large Ventral Hernia—Strangulation—Resection of* 9 *ft. of Intestine.*—Female, aged 50, admitted to the Royal Victoria Infirmary, 14/10/10.

History.—Three and a half years previously she had been operated on for an appendix abscess, which was opened and drained. After the wound healed she developed a hernia through it, which had gradually increased in size, but caused no inconvenience til

two days ago, when pain in her abdomen commenced at 8 P.M. Six hours later she vomited. Since then the pain and vomiting had continued, she had much rumbling of wind, and had passed no flatus or fæces.

On admission the patient was an enormously stout woman ; she looked fairly well ; tongue moist and furred ; pulse, 84 ; temperature, 100° F.

The abdomen was swollen. A large ventral hernia on the right side under a scar was tense, tender, and irreducible. Part of it was softer than the rest.

Diagnosis.—Strangulated ventral hernia causing acute intestinal obstruction. An operation was performed by Mr Richardson, who found the tumour about equal to two footballs in size, the sac multi-locular and containing some tightly strangulated intestine. The strangulation was relieved, but it was obviously impossible to replace the contents of the sac in the abdomen, and the skin was sutured over them. The wound suppurated, a fæcal fistula developed, and an attempt was made unsuccessfully to close this on 9/11/10.

Operation—20/11/10. *Mr Morison.*—A large elliptical incision was made enclosing the fistula, the edges were closed by sutures, and the sac opened. The underlying intestinal coils were firmly adherent to the skin and to each other, and great difficulty was experienced in separating them and defining the opening of the sac. Eventually 9 ft. of small intestine were resected, the lower point of division being just above the ileo-cæcal valve. An end-to-end anastomosis was made with a glass bobbin and by invagination, the sac was excised, its neck sewn up, and the abdominal wall was closed in separate layers, with a tube under the skin for drainage.

The patient made a good recovery, but on 9/12/10 (3 weeks after operation) it is noted that " her bowels are moved 5 times daily."

20/12/10.—" Patient made a good recovery. The diarrhœa is much better." Recovered.

Subsequent Note.—This patient died at home 2 months later as the result of persistent diarrhœa.

CASE .—*Multiple Malignant Strictures of Small and Large Intestine.*—Male, aged 39, admitted to private hospital, 18/5/10

History.—Five weeks previously he had an attack of acute intestinal obstruction, but it only laid him up for 6 days. Since then he had suffered from pain, flatulence, and occasional vomiting

till 24 hours before admission, when he had another acute attack. Fourteen years ago he had phthisis pulmonalis.

On admission he was in very poor condition; pulse, 95; temperature, 99.2° F. All the signs and symptoms of acute intestinal obstruction were present, with free fluid in the abdomen.

Diagnosis.—Acute intestinal obstruction due to tuberculous peritonitis.

Operation.—The abdomen was opened by an oblique incision in the right iliac fossa and a quantity of blood-stained fluid escaped. A malignant stricture was found in the end of the ileum, with the gut on its proximal side much distended and the cæcum empty and collapsed. The entire visible and palpable peritoneum was studded with malignant growths and also the omentum, a portion of which was removed for microscopical examination. A lateral anastomosis was made between the ileum above the stricture and the ascending colon and the abdomen was closed.

Progress.—For the first two days after operation the urgent symptoms were relieved, but he was never well. Later the symptoms of obstruction returned, and he died 5 days after the operation (23 5 10).

Post-mortem examination showed malignant deposits studded everywhere in the abdomen. There were several on the small intestine, causing a certain amount of stricture. There was a tight stricture of the hepatic flexure of the colon, which was obviously the cause of recurrence of the obstruction. There was septic peritonitis.

Microscopic Report.—"Columnar-celled cancer, with a tendency to colloidal degeneration."

CASE .—*Spheroidal-celled Carcinoma of the Ascending Colon with Ovarian Cyst.*—M. J. G., aged 62, married. Admitted 6 4 10. Died 16 4 10.

Patient gave a history of many years' duration of attacks of indigestion, accompanied by excessive wind and vomiting. Three weeks ago she began to have pains in the abdomen. Four days ago, after cycling in the evening she was seized with severe griping pain in the abdomen; castor oil gave a good evacuation of the bowels and relieved her somewhat, but she did not sleep well at night. The following day she was fairly well, but sent for the doctor in the evening on account of a feeling of heaviness in the stomach. She vomited repeatedly the next two days, the vomiting

being bilious and not fæcal. On the evening before admission she felt much better and remained so till the day after, *i.e.* 7/4/10, when she vomited again. Rumblings were present with the attack, and flatus was passed.

On admission, patient was of a sallow complexion, and looked very weak, thin, and emaciated; temperature, normal; pulse, 90.

Abdomen.—On inspection distension present, suggesting small intestine. On the right side opposite the umbilicus there was felt a hard nodule, not very tender and not movable. A suggested diagnosis was "impacted gall-stone" in the small intestine, for the following reasons:—

1. The long history of "indigestion," with wind and vomiting.
2. Since present onset her tendency to have periods of improvement.
3. The feeling of the tumour.
4. The limitation of distension, etc., to small intestine.

Peristalsis was visible, of the oblique ladder pattern. The patient was too ill for a radical operation and not ill enough for enterostomy.

10/4/10.—Patient improved, bowels moved. A tumour was now felt at the same site as previously, very hard and nodular, and quite fixed. Previous diagnosis negatived. Per vaginam an ovarian cyst was felt, the size of a fœtal head, uncomplicated.

Present Diagnosis.—Carcinoma of the ascending colon, malignant mass secondary in the ovarian tumour.

12/4/10.—Symptoms of obstruction have increased; patient very ill.

Operation—13/4/10. *Mr Morison.*—Long oblique incision, as for appendix abscess, extending well back into the loin. Abdomen opened. The tumour appeared to be a malignant growth of the ileo-cæcal valve, with many large and secondary glands in the mesentery extending up to the duodenum. The outer reflection of peritoneum from the cæcum and ascending colon on to the posterior belly wall was now divided and this part of the large intestine easily separated and brought forward. The ileum was divided about 6 ins. from its termination and then the distal part of the ileum, the cæcum, ascending colon, and $\frac{1}{4}$ of the transverse colon were removed. The procedure was most difficult owing to the huge glands in the mesentery, extending to and lying on the third part of the duodenum. The ileo-colic artery was divided close to its origin from the superior mesenteric, the vein

being clipped at about the same spot. There was some hæmorrhage from slipping of the ligature. The glands were the size of walnuts, hard and surely malignant. The cut end of the transverse colon was closed in the usual way, but the end of the ileum was brought out of the wound posteriorly and a Paul's tube tied in. The abdominal wound was sutured in layers.

Pathology.—The growth proved to be arising in the ascending colon, having spread into the cæcum, but not having involved the ileo-cæcal valve. The glands, extending right up to the duodenum, appeared to be infiltrated with growth.

Microscopic Report.—"The lymph gland has been almost entirely replaced by a diffuse carcinomatous deposit; appearances suggest a rapidly advancing growth of spheroidal type, almost encephaloid in character. The tumour is a rapidly growing columnar-celled cancer, showing a tendency to diffuse infiltration, with cells of a more spheroidal type."

Subsequently patient suffered from shock, and as Paul's tube did not drain well, Mr Morison performed enterostomy higher in the intestine under anæsthesia, but no fluid or flatus was passed. She died 15/4/10, in spite of frequent stomach lavage, oxygen, and saline.

Post-mortem.—No sepsis; no dilatation of the stomach; operation sites healthy; small intestine still distended. A single enlarged hard gland the size of a filbert was found in the aorta. A large ovarian cyst the size of a cocoa-nut was found in the pelvis; it was not malignant.

CASE .—*Adenomatous Polypus of Small Intestine with Chronic Intussusception.*—A male, aged 64, admitted to the Royal Victoria Infirmary, 3/11/10.

History.—Three weeks ago the patient, previously quite well, was suddenly seized with a sharp pain in his stomach half an hour after his dinner. An hour later he vomited matter of a dark-brown colour. This did not relieve the pain. A bowel motion which he had the same evening was very dark in colour. The pain had lasted off and on till his admission. It was worst after meals and woke him up at nights. Vomiting had not been frequent. His bowels had acted regularly and were not constipated. On the morning of admission he had retention of urine, which was relieved by a catheter.

On admission he looked a robust man somewhat reduced by pain; temperature and pulse were normal.

Abdomen.—There were definite rumbling sounds heard in the abdomen and active peristaltic movements of the small intestine type were visible. A tumour could be felt to form and harden intermittently close to the right of the umbilicus. Both flanks were dull on percussion.

Diagnosis lay between a malignant growth of the intestine and chronic intussusception, possibly both.

Operation—4/11/10.—The abdomen was opened in the middle line after excision of the umbilicus. The fluid escaped; the small intestine was large, thick and congested. An intussusception 5 ins. long was found in the small intestine close to the ileo-cæcal junction. On reducing it a tumour about the size of a walnut was felt in the lumen of the bowel. Enterostomy was performed and the polypus was excised after ligaturing its pedicle. The intestinal wound was closed by an inner layer of through-and-through catgut sutures and an outer layer of silk Lembert sutures and the abdomen by 3 tiers of sutures. Recovered.

Report of Pathologist on Polypus.—"This appears to be a simple adenomatous polypus. The fibrous core shows a considerable quantity of unstripped muscle; it is very vascular, and shows œdematous change. There is some inflammatory change superadded."

Case .—*Malignant Papilloma of Sigmoid—Resection.*—A male, aged 58, admitted to Royal Victoria Infirmary, 11/11/10.

History.—The patient was operated on for hæmorrhoids 5 years ago, and for the last 4½ years had been treated for mucous colitis. With his stools, which were usually looser but sometimes consisted of round hard masses, he had passed slime and blood. He often had a twitching pain before defæcation and, after it, a feeling that his bowel had not been properly emptied, but never a severe pain, only a bearing down. When flatus was passed mucus often escaped involuntarily at the same time.

On admission he looked well and had a normal pulse and temperature.

Abdominal and *rectal* examination were negative in result.

With the sigmoidoscope (Mr Hamilton Drummond) a small polypus on the right rectal wall and, 7 ins. from the anus, and out of digital reach, a large soft flattened papillomatous growth, which bled readily, were seen.

Operation—17/11/10.—The abdomen was opened by an oblique

incision in the left iliac fossa and the growth was felt in the lower part of the pelvic colon. The lower half of the sigmoid with its mesentery was excised and an end-to-end anastomosis by the invagination method was performed between the remainder of the sigmoid above and the rectum below. The abdominal wound was closed in 3 layers. Recovered.

Pathological Report.—"This appears to be a papillary adenoma. Owing to the complicated papillomatous arrangement it is difficult to determine the absence of infiltration into the muscularis mucosæ, but so far as can be seen at present the condition does not appear to be malignant."

Note.—I made a macroscopic section and found the bowel wall infiltrated with growth, and have no doubt of its malignancy.—R. M.

CASE .—*Ileo-cæcal Tuberculosis mistaken for Appendix Abscess—Chronic Intestinal Obstruction—Resection—Enterostomy——Acute Intestinal Obstruction and a Second Enterostomy—Excision of Involved Intestine and Anastomosis.*—Male, aged 30, admitted to the Royal Victoria Infirmary, 24/4/10.

History.—For the past 3 weeks the patient had had trouble in his bowels—griping pains and much rumbling. Four days before admission he was suddenly seized with very severe pain at 3 P.M. The pain was intermittent and was associated with frequent vomiting and much rumbling. He was able to pass flatus. His bowels had moved daily. During his illness he had heavy night sweats and had lost about 1 stone in weight.

On admission he looked feeble and ill, but his acute symptoms had subsided. His tongue was furred; pulse, 90; temperature, 99° F.

Abdomen.—There was no rigidity. A large hard mass, slightly tender, was found in the right iliac fossa with a smooth surface, except for a small hard nodule which projected from its upper and inner aspect; its edge was fairly well defined and the whole swelling was slightly movable. During palpation gurglings could be heard and felt in its neighbourhood. Percussion over the lump showed diminished, not absent, resonance.

Diagnosis.—Appendix abscess of long duration with dense adhesions.

Operation—26/4/10.—The abdomen was opened by a long oblique incision in the right iliac fossa. The mass proved to be no abscess. The bowel and peritoneum in the right iliac fossa were

enormously thickened and infiltrated and so adherent that the appendix could not be isolated. It was impossible to say whether the condition was the result of growth or inflammation, and it was decided to remove the whole of it. The Jameson-Dobson operation of removal of the whole ileo-colic distribution was performed and the cut end of the colon was closed by sutures. On account of his poor condition a Paul's tube was tied in the divided end of the ileum and the wound was closed in layers round this. For some days his progress was satisfactory, then he began to develop abdominal pain and to vomit, with increased peristalsis and rumbling; the discharge from the ileum ceased and it was clear he had acute intestinal obstruction.

Second Operation—7/5/10, by *Mr Richardson*.—The abdomen was opened by a median incision and the obstruction was found to be due to constriction of the gut by the healing of the wound. The constriction and gut were divided and the obstruction was relieved. Subsequently the patient improved slowly, but refused further surgical help, and decided to go home to the country with his enterostomy wound discharging and the surrounding skin in an excoriated condition.

Pathology.—The specimen consisted of the last 6 ins. of the ileum, the cæcum, the ascending colon, and ileo-colic mesentery. On section the lumen of the cæcum up to the ileo-cæcal valve was almost obliterated by a dense white fibrous cicatricial mass. The mucous membrane appeared to be normal but was thrown into polypoid folds by the fibrosing submucous tissue. The end of the ileum was prolapsed for about 1 in. into the cæcum, so as to look like the uterine cervix. (This appearance is common in tubercle and cancer of this region.—R. M.) Enlarged glands were present throughout the mesentery.

Microscopic Report, Professor MacDonald.—"*Gland*.—No evidence of malignant disease or tubercle. Appears to be of simple inflammatory origin. *Growth?*—Hyperplastic thickening of all the coats of the intestine, with marked lymphocytic infiltration of all the coats. The condition is probably tuberculous though there is no distinct evidence of it. There is no evidence of tumour or growth."

Readmitted, 16/9/10.

The patient's condition was much improved, and he had gained considerable weight since going out. The median wound was entirely healed; from the iliac one the mucous membrane of the

bowel was protruding. All fæces had been passed through this since his first operation.

Third Operation — 22/9/10.—The old median scar (Mr Richardson's) was excised, and it was surprising to see so few (none but necessary) adhesions in the peritoneal cavity. A lateral anastomosis was made between the lower ileum and the right transverse colon with a through-and-through inner layer of catgut sutures and an outer of silk, then the ileum was divided transversely and its distal end sewn up proximal to the anastomosis. The mesentery of the isolated bit of ileum was next tied off and cut, a pair of forceps was passed up through the enterostomy opening, and the bowel, turned inside out, was drawn through the enterostomy opening. The edges of the median incision were next closed, and then the inverted bowel and the edges of the oblique iliac wound were excised. When this had been done it was found that the edges of the iliac wound could not be drawn together, and that a gap of at least 1 in. remained with the tightest suture that could be applied. This was considered to be due to contraction of the divided muscles, and it was thought that the traction of temporary tight-thick sutures would in a few days overcome the retraction and allow of approximation. The right iliac fossa was consequently packed with gauze and six thick sutures with a good bite were passed through the edges and whole thickness of the wound and tied firmly in a bow knot over the gauze. Four days later (26/9/10) the gauze was taken out and the silk sutures were tightened up, bringing the wound edges readily into apposition. He was discharged in excellent condition with the wound healed except for small superficial granulations 3 weeks later (10/10/10). Recovered.

Case .—*Hyperplastic Tuberculosis of the Cæcum—Excision.*—Female, aged 20, single, was admitted to private hospital, 8/10/10.

History.—She was quite well till 2 years ago. Eighteen months ago she was operated on for appendicitis. Five weeks after the operation a sinus formed in the wound and had continuously discharged fæcal matter since. She had had much cramping pain in the right iliac fossa with wind rumblings, and had to sleep with a hot bottle on the side.

On admission the patient was bright, intelligent, and fairly healthy looking. No disease was found except that noted.

Abdomen.—There was a keloid scar in the right iliac fossa with

two fistulæ posteriorly, from which sero-pus was discharging. There was a tender resistant mass to the right of the umbilicus.

Diagnosis.—Ileo-cæcal tuberculosis.

Operation—12/10/10.—The old scar was excised and the abdomen was opened by a long oblique incision in the right iliac fossa after dividing the skin in an ellipse round the fistulæ, turning it over a dossil of gauze, and suturing it. A tangled adherent mass was found in the right iliac fossa involving the ileum, the cæcum, and the transverse colon which had been dragged down to it. The vermiform appendix was lying below, separate, free, and apparently healthy. After mobilisation of the mass by division of its outer and upper peritoneal reflections, the lower 6 ins. of the ileum, the cæcum and vermiform appendix, the ascending and the right half of the involved transverse colon and their mesentery were excised, and the ends of the colon and ileum were closed, a lateral anastomosis being made between the ileum and the left half of the transverse colon by a Murphy button. During the operation the patient, although apparently unhurt, had intravenous transfusion of 2 pints of normal saline. Recovered.

Pathology.—The mesentery contained many enlarged caseating glands, some of which were calcareous. The bowel wall was much thickened—a typical example of hyperplastic tuberculosis—and the ileo-cæcal opening constricted.

Microscopic Report.—"The section is typical of hyperplastic tuberculosis."

Case .—*Hyperplastic Tuberculosis of the Cæcum.*—T. R., aged 48, married. Admitted 18/3/10. Discharged 13/4/10.

Patient gave a long history of abdominal trouble. Ten years ago he had an attack of what he called "catarrh," accompanied by diarrhœa and sickness, and associated with swelling of the abdomen. With this attack he was laid up in bed for 3 weeks, and he had frequent subsequent similar attacks, lasting a few days. Two years ago he was awakened during the night by a sudden severe pain, spread diffusely over the abdomen, which lasted 5 hours. The pain was accompanied by rumblings and diarrhœa, and he vomited repeatedly, this latter lasting 24 hours. His motions during the attack were black, and the doctor stated this was not due to medicine. Heat relieved the pain somewhat. He was in bed for 3 weeks and had no return of the pain, but his right side was very tender, and he was kept on light diet for some time. He

remained well after this till a month ago, when he had a slight similar attack and another a fortnight ago, which only kept him in bed for 1 day. On the day before admission he was again seized at night by a very severe pain across the abdomen, lasting a few hours and gradually subsiding, so that by the day after admission he was unable to feel it. He was sick and vomited several times the day following the onset, but the vomitus was not fæcal. The pain was never localised. As a general rule his bowels had to be regulated by aperients; he had had no urinary symptoms.

On admission patient looked in fair condition, though thin; temperature, 99.2° F.; pulse, 66; chest, clear.

Abdomen.—Thin belly wall; no suggestion of abnormality on inspection. On palpation a definite tumour the size of a hen's egg could be felt in the right iliac fossa; it was hard in consistency, its surface was rounded, the upper edge was well defined though it was more indistinct below, and it was fairly well fixed down to the iliac fossa, while the anterior belly wall was freely movable over it. Gurgling was demonstrated above the tumour but not over it, and the mass was quite separate from the kidney.

Per Rectum.—Nothing abnormal was felt in the lumen, but high up on the right side an enlarged gland the size of a hazel-nut was felt.

Diagnosis.—Tuberculosis cæcum—chronic appendix.

Operation—23/3/10. *Mr Morison.*—Long oblique incision in the right iliac fossa prolonged well back into the loin. The tumour was exposed and found to be a chronic inflammatory mass at the caput cæci, with a firm matting of adhesion to the middle of the transverse colon. The appendix was found buried in the mass and was thought to be the origin of the trouble, being firmly adherent to the cæcum and communicating with it by a large hole which would admit the little finger. The appendix was removed, was found to be about 2 ins. long, and was little more than a fibrous band. After its removal there was still a large hard fibrotic thickening in the cæcal wall, and, owing to the doubt entertained as to its nature, it was considered the proper treatment to remove it. The ileum was divided about 5 ins. from its termination between clamps by the cautery, and the cut end was closed in the usual way by through-and-through catgut and silk lemberts. The ascending colon was divided immediately above the cæcum and dealt with in a similar manner. A lateral anastomosis between the ileum and ascending colon was now performed by direct suture—through-and-through

catgut sutures covered in by silk lemberts. A small opening was purposely made to imitate the ileo-cæcal valve. Abdominal wound closed in layers.

Specimen.—This showed thickening of the cæcal wall, but on inverting the latter there was no definite evidence of growth (tubercle or cancer) in the lumen of the gut. In the mesentery of the cæcum was a hard calcareous gland, the only real evidence of tuberculosis. There was slight stricture of the ileo-cæcal orifice, but the valve showed no change.

Microscopic Report.—Hyperplastic tuberculosis. Peritoneal coat is thickened and very vascular ; muscular and submucous coats are markedly thickened and show diffuse fibrosis, also numerous lymphocytic foci with caseous centres and a few typical giant cells.

12/4/10.—Healed by first intention ; good recovery. Going home.

Case .—*Ileo-cæcal Tuberculosis ?—Acute Intestinal Obstruction —First Operation—Lateral Anastomosis. Second Operation—Excision of Bowel.*—A miner, aged 18, admitted to Royal Victoria Infirmary, 27/6/10.

History.—Two years previously he had an attack of pain in the right iliac fossa associated with vomiting, diarrhœa, and the passage of bright red blood. At this time he was in a hospital for 3 months, and during his stay there he had night sweats, and for 6 weeks his temperature varied from 101° F. to 106° F. Subsequently he worked as a miner, but had frequently recurring attacks of diarrhœa unaccompanied by bleeding. During the last two months he had lost weight from continual diarrhœa, his bowels being moved 8 or 10 times daily. For the last month he had been unable to work from abdominal distension, pain and rumblings.

His mother died of consumption.

On admission he was not looking ill, and nothing wrong was found except as noted.

Abdomen.—This was considerably distended, and peristalsis of the oblique ladder pattern, characteristic of obstruction low in the small intestine, was present. No mass was palpable, and there was no evidence of free fluid.

Diagnosis.—Intestinal obstruction due to ileo-cæcal tuberculosis.

Operation—28/6/10.—The abdomen was opened in the median line below the umbilicus. The whole of the ileo-colic group of glands was enlarged and hard, and there was a mass of adhesions

round the cæcum. A lateral anastomosis was performed between the lower end of the ileum and the transverse colon, and the abdominal wound was closed in layers.

For 12 days after the operation he was free from his old pain and then had a mild attack of intestinal obstruction. This was followed by others of a similar nature, though his general condition had much improved.

Second Operation—9/8/10.—A large firm mass was now felt under the anæsthetic in the right iliac fossa. The old scar was keloid. The abdomen was opened by excision of the umbilicus and the scar. Seven inches of the ileum distal to the anastomosis, the cæcum, vermiform appendix, ascending colon, and the transverse proximal to the anastomosis with their mesenteries were excised, the divided ends of the ileum and the transverse colon sutured, and the abdomen closed by 3 tier sutures. Recovered.

Pathology.—A band of omentum was adherent to the ascending colon. (It seemed likely that this was the cause of the obstruction.) The whole of the excised gut was hypertrophied and œdematous, and some tuberculosis was present on the outer surface of the cæcum. There was thickening of the ileo-cæcal valve but no obstruction.

Microscopic Report.—"The wall of the intestine is swollen and œdematous, but there is no evidence of inflammatory thickening. The mucosa is especially swollen and thrown into thick folds, and there is an excessive formation of mucus in the lining cells. The thickening is largely the result of œdema. There is no evidence of tubercle or new growth."

Case .—*Carcinoma of Caput Cæci intussuscepted into the Ascending Colon.*—S. A., aged 64, widow. Admitted 7/4/10. Discharged 2/5/10.

Patient admitted with a lump in her right side. She first noticed the lump 7 days ago. She had great discomfort and a feeling of fulness, but no pain. On the day before admission pain commenced across the abdomen, going down to the lump, gradually increasing in severity as the day went on. Medicine only relieved her temporarily, the pain recurring at intervals afterwards. Since the commencement of her illness she had noticed rumblings, not loud but frequent. During the last 3 months she had been losing flesh and her appetite had been very poor. Her mother died of cancer of the breast.

On Admission.—General condition: thin, pale, small, feeble woman; temperature, 98° F.; pulse, 80.

Local.—In the right side there was a soft cystic-like mass under the bowel; it moved freely on manipulation, but not much with respiration. The whole mass was indefinite, and the characters difficult of ascertainment. It was somewhat tender. Per vaginam a definite tumour rounded and tender in the right iliac fossa and so was in close contact with the uterus.

Diagnosis.—Carcinoma of ascending colon or ovarian cyst (pedicle? twisted).

Operation — 12/4/10. *Mr Morison.*—Median infra-umbilical incision; abdomen opened. The ileum was found intussuscepted into the ascending colon, the apex being formed by the cæcum with a growth. The pelvis was found clear, thus putting out of count the possibility of ovarian tumour. The lower part of the ileum, the cæcum, and ascending colon were now excised, being divided between clamps by thermo-cautery. The part of the mesentery in the ileo-colic distribution was also removed to clear out the glands and lymphatics. The cut ends of the gut were closed in the usual way by continuous through-and-through catgut and overlying silk lemberts. Lateral anastomosis was now proceeded with by simple suture between the lower end of the ileum and the transverse colon. The silk lemberts were interrupted. The anastomotic opening was about 1½ ins. long. Abdominal wound was closed in layers in the usual way.

Pathology.—The growth appeared to be in the caput cæci and not ileo-cæcal valve, which ultimately proved to be so, it arising about the base of the appendix, which could not be found. The growth was sessile and the size of a walnut.

Microscopic Reports.—"The tumour itself is a columnar-celled carcinoma, widely infiltrating beyond the muscularis muscosæ. Gland from mesentery shows chronic inflammatory change, marked endothelial proliferation, but no evidence of malignant deposit."

2/5/10.—Patient made an excellent recovery and left hospital healed.

Case .—*Carcinoma of Ascending Colon—Death from Acute Dilatation of the Stomach.*—J. F., aged 43, married. Admitted 14/3/10. Died 17/3/10.

Admitted complaining of a lump in the right side. History of 15 months' abdominal trouble. He had good health previously,

but since this time he had had attacks of severe pain across his lower abdomen almost every day, coming on suddenly and going off quite as suddenly. They came on at any time, having no relation to food or rest. He had never been sick or vomited, there had been no intestinal rumblings, and the pain was not relieved by defæcation or micturition, nor did change of position affect it. He had never been jaundiced, and had had no diarrhœa. He had lost 1 stone in weight during the last 2 months, his appetite had gradually become worse, and recently he had not slept well. Patient's father died of "cancer of the bowel."

On admission patient looked in fairly good condition; temperature, 98.8° F.; pulse, 100.

Local.—A hard nodular mass was felt to the right and a little below the level of the umbilicus. It was well defined and freely movable by manipulation, and could be moved into the flank. It moved slightly with respiration, but it could be prevented from so doing by grasping it bimanually. It was not tender, though he held himself somewhat rigid, thus rendering palpation slightly more difficult than usual. Nothing else abnormal could be detected in the abdomen.

Per Rectum.—Nothing abnormal felt. Patient had an occupation kyphosis and slight scoliosis—no rigidity of spine.

Diagnosis.—Appendix abscess.

Operation — 16/3/10. *Mr Morison.* — Resection of ileum, cæcum, and ascending colon (Jameson-Dobson) for a growth in the ascending colon; lateral anastomosis between stump of ileum and transverse colon by direct suture. No secondary deposits on peritoneum or liver. Patient stood the operation well.

After-Progress.—Patient appeared well for the first 40 hours, when he collapsed and presented signs of acute dilatation of the stomach. On washing it out it contained about ½ a pailful of dirty blackish fluid. It was washed out 2 or 3 times subsequently, on each occasion a large quantity of fluid being removed. He did not pick up, and died 3 days from the operation.

Post-mortem.—Belly opened; no signs of any sepsis. All in connection with the operation perfect. The stomach was so large and lax as to occupy the greater part of the abdomen, the pyloric end of the first part of the duodenum containing many submucous hæmorrhages; the duodenum and 2 ft. of the adjoining jejunum were also very much dilated.

Microscopic Report of Gland Removed.—Chronic inflammatory changes; no evidence of malignant deposit.

Growth.—Carcinoma of columnar-celled type.

CASE .—*Carcinoma of Ascending Colon—Injury 3 Years Previous—No Bowel Symptoms.*—W. H., aged 59. Admitted 4/3/10. Death 29/3/10.

Admitted complaining of pain in the back and across the bowels. Ascribes his trouble, three years ago, to a fall of stone down the pit. His back was injured and he had loss of power and sensation in his legs, and marked hæmaturia for 3 or 4 days. He had never been fit since, and had been in receipt of compensation practically continuously up to the present. For the past 2½ years he had complained of short sharp pains in the right loin, with pain and swelling in the abdomen, the loin pain radiating into the inguinal region, but not into the scrotum or thigh. It had become worse, and lately he had complained of soreness in the back when touched there. Some days patient passed a great deal of clear urine, while on others only a little, with brick-red deposit.

Frequency.—None during the day; had to get up 2 or 3 times during the night.

Pain.—Had some at the end of micturition.

Stream.—Some difficulty in starting, and the stream often stopped and recommenced; size and force usually good.

Urine.—Had noticed no blood, gravel, or "matter" in urine. Patient had lost at least 1 stone in weight recently. He had had no vomiting and was constipated; troubled with flatulent rumblings.

On admission patient very anæmic; temperature, 98.2° F.; pulse, 100.

Abdomen Inspection.—A slight bulging was seen in the right loin, somewhat below the level of the umbilicus.

Palpation.—A large hard tense tumour was felt in the right iliocostal space, rather lower than usual for kidney. It was somewhat movable bimanually, and moved with respiration. It was slightly tender.

Percussion.—Loin was dull; tumour had resonance in front of it. No other abnormality found.

Urine.—Sp. gr. 1010.

Acid.—No albumen or sugar.

X-Ray of Right Kidney.—No abnormal shadow shown.

Cystoscopy—Bladder normal. An attempt at catheterisation of the ureters failed from hæmorrhage into the bladder.

Diagnosis.—Carcinoma of kidney (though rather low in situation).

Operation—15/3/10. *Mr Morison.*—Incision through right rectus muscle to enable the opposite kidney to be examined. On opening the abdomen the mass was found to be a large malignant growth of the upper part of the ascending colon lying on the kidney. The ascending colon with the growth was mobilised by dividing the lateral reflection of the peritoneum on the outer side of the bowel and then the lower part of the ileum for about 6 ins., and the whole ascending and half the transverse colon with the whole of the mesentery in the distribution of the ileo-colic artery were resected with their glands and lymphatics (Jameson and Dobson operation), the gut being divided with the thermo-cautery, and the ends closed by through-and-through catgut and super-imposed silk lemberts. A lateral anastomosis was performed by simple continuous suture between the ileum and transverse colon. Belly closed in layers after insertion of drainage tube through the loin. Patient transfused during the operation.

Microscopic Report of Growth.—" Columnar-celled carcinoma."

Four days after operation patient developed a fæcal fistula, and went steadily back. He died on the 29/3/10—14 days after operation.

Post-mortem.—Showed enormous dilatation of the stomach. Much pus and fæcal matter were found at the site of operation. The anastomosis could not be demonstrated, and the gut was open at that site, the sutures having evidently given way. The distal ends of both parts of the bowel were closed and showed no evidence of yielding—there was so much matting and dirt that it was difficult to obtain a clear view. The liver had two small nodules in it, which were sent for microscopic report. Kidneys and other organs healthy.

Microscopic Report of Nodule from Liver.—" Columnar-celled carcinoma ; undergoing extensive colloidal degeneration."

Case .—*Malignant Gall-Bladder Adherent to and Perforating the Hepatic Flexure of the Colon, and Causing Chronic Intestinal Obstruction.*—Female, aged 61, married, admitted to private hospital, 11/7/10.

History.—Four months previous to admission she began to be troubled by abdominal pain and rumblings, and began to lose flesh rapidly. The pains were similar to those produced by taking

strong purgative medicine, and had kept her much awake at night. The rumblings were so loud as to be heard by anyone near. She had great difficulty in getting the bowels to move. Had never been sick or had diarrhœa, and had no appetite. For 20 years she had suffered from " flatulence and spasms " in the stomach, coming on in attacks.

On admission she looked fairly well, but was thin. Tongue clean and moist ; teeth, false ; pulse, 108 ; temperature, normal.

Abdomen.—In the right iliac fossa a large hard nodular mass was found. Its lower and lateral margins were well defined, but its upper limit could not be satisfactorily felt. It moved slightly downwards on deep inspiration, was not firmly fixed, and did not extend into the loin behind. There was no evidence of free fluid in the peritoneum, and no palpable enlargement of the liver or of the supraclavicular glands.

Diagnosis.—Carcinoma of the ascending colon, causing chronic intestinal obstruction.

Operation—14/7/10.—Under the anæsthetic the tumour could be seen moving very freely with respiration. An oblique incision opened the abdomen over it and exposed the omentum lying over and firmly fixed to the tumour, suggesting an error in diagnosis. On separating the omentum the peritoneal cavity was packed off and a knife was thrust into the outer aspect of the tumour, which was now clearly seen to be an enlarged gall-bladder. About 4 ozs. of pus and a number of gall-stones escaped. The gall-bladder, which was very adherent, a tongue-shaped projection of liver, and a portion of the right lobe to which it was attached were excised with great difficulty, a perforation between the gall-bladder and the hepatic flexure of the colon being exposed and sutured. The abdominal wound was closed in layers without drainage. Recovered.

Pathology.—The gall-bladder had very thick walls calcareous in parts. It presented multiple holes where it had perforated into the colon and the liver. The calculi varied in size from a blackbird's egg to a millet seed, the largest being impacted in the neck of the gall-bladder. All were white on the surface from a coating of cholesterin.

Microscopically a section showed typical columnar-celled cancer.

Case .—*Carcinoma of Transverse Colon—Excision and Lateral Anastomosis.*—A. H., aged 44. Admitted 8/4/10. Discharged 29/4/10.

Patient gave a 7 weeks' history of bowel trouble, though she stated that $2\frac{1}{2}$ years ago she had a large discharge of "corruption" from the bowels—blood and other material. This did not recur, and she had been free from symptoms till 7 weeks ago. Since then she had had abdominal pain, of an intermittent character, lasting only for a short period at each attack and associated with rumblings of wind, which were quite loud. She had passed no flatus recently, but for how long she was unable to say. She had noticed that the bowels swelled up and lumps appeared, which came and went with the rumblings. Her bowels had for the most part been constipated, but she had one attack of diarrhœa lasting 2 days, followed by constipation. She had passed no blood. She had lost a great deal of flesh lately, and had been unable to rest for pain.

On admission the patient was much emaciated; tongue, dry and furred; temperature and pulse, normal; chest, clear.

Local.—Abdomen flaccid, but some distension present. A large mass was seem to rise in the upper part of the abdomen, with peristalsis from right to left, ceasing at the left costal border, like the transverse colon. This occurred intermittently. On palpation no tenderness or rigidity were present, but a mass was felt in the left hypochondrium, hard, nodular, fairly well defined, and freely movable. It did not move with respiration. The portion of gut which was above described as exhibiting excessive peristalsis, and which was felt to harden, terminated at this mass. The liver was felt below the costal margin, but no nodules were demonstrable on its surface. No free fluid in the abdomen.

Per Rectum.—Some ballooning was present, but no evidence of growth. No glands were felt in the left posterior triangle of the neck, or any secondary deposits in the pelvis.

Diagnosis.—Carcinoma of the colon in the neighbourhood of the splenic flexure, with obstruction of the bowel from stricture at this site.

Operation—14 4 10. *Mr Morison.*—Median supra-umbilical incision; abdomen opened. A large freely movable malignant growth was found in the transverse colon, with secondary glands in the meso-colon adjacent. The growth was resected with several inches of bowel on either side and the associated meso-colon. The bowel was clamped and divided by the thermo-cautery, and the ends closed by through-and-through sutures of catgut with overlying silk lemberts. A lateral colico-colic anastomosis was now

performed by simple suture, the silk lemberts being interrupted. Belly closed in layers.

Subsequent.—Patient very ill for the first few days, but made an uninterrupted recovery, and left hospital with wound healed, and very well, 29 4 10.

Microscopic Report of Growth.—" Columnar-celled carcinoma."

Case .—*Inoperable Carcinoma of Splenic Flexure of Colon.*—A female, aged 65, admitted to the Royal Victoria Infirmary, 20 6 10, with attacks of repeated intestinal obstruction of 3 months' duration.

Operation showed an adherent growth of the splenic flexure with extensive glandular involvement. Permanent colostomy in transverse colon. Recovery.

Case .—*Intestinal Obstruction from Malignant Growth in Sigmoid Colon—Three Operations.* 1. *Cœcostomy for Relief of Obstruction.* 2. *Excision of Growth.* 3. *Closure of Cœcostomy.*—G. B., age 62, engine-driver. Admitted 27 11 09. Discharged 27 1 10.

Patient admitted with extrene constipation and swelling of the abdomen. Six months previously his illness commenced with an attack of severe abdominal pain, during which he passed no flatus or fæces, but had some vomiting; subsequently the constipation gave place to diarrhœa, and he obtained relief. Since that time he had had frequent attacks of pain in the abdomen, accompanied by rumblings of wind, and his abdomen had swelled up very considerably. He had been troubled much with hiccough and eructations of gas, but he had passed little flatus. Constipation had been marked since the first attack described, his motions being very small in calibre and in the form of pellets, while he had occasionally passed slimy material but no blood. He had lost a good deal of weight since the onset.

On admission patient was very ill, being emaciated and cachectic; pulse regular but soft; temperature, 98.6°; chest, clear.

Local.—Abdomen uniformly distended, flanks as well as anteriorly. The cæcum and ascending colon were easily recognised outlined through the belly wall, the contour of the latter varying in distinctness from time to time as they contracted. On palpation the colon could be easily felt when it contracted; this occurred intermittently. Succussion could be obtained in the cæcum, but no tumour or other mass could be felt in the abdomen. On percus-

sion the whole belly was tympanitic, without flank dulness, the liver being displaced up above the costal margin. No gland was felt in the left posterior triangle at the neck.

Per Rectum.—The lower portion of the gut was felt ballooned, and the examining fingers could just touch a hard nodular mass, like a uterine cervix, high up in the bowel; its relations could not be made out, *i.e.* with regard to its fixity and extent, on account of its height.

Diagnosis.—Carcinoma of the upper part of the rectum, with chronic intestinal obstruction.

Operation—1 12 09. *Mr Richardson.*—Small oblique incision made in the right iliac fossa. Abdomen opened and a large quantity of free fluid escaped. The cæcum was pulled out and sutured round to the wound margin. Bowel wall incised and Paul's tube introduced and fixed with silk. Distension relieved.

Subsequent.—Tube sloughed off on the 5th day; cæcostomy looked well.

Second Operation—18 12 09. *Mr Morison.*—Median infraumbilical incision; abdomen opened. On examination a malignant growth was felt in the first part of the rectum, but no enlarged glands or deposits in the liver could be palpated. A small (1½ ins.) incision was made in the left iliac fossa, opening into the peritoneal cavity. The sigmoid was divided above the growth, the upper end being closed and fixed in the left inguinal incision for permanent colostomy. The superior hæmorrhoidal artery was ligatured with the meso-rectum, and the growth with the sigmoid flexure and upper part of rectum resected. The cut lower end of the rectum was closed with 2 layers of sutures. The median laparotomy incision was closed in layers. Two days later the closed end of the sigmoid projecting through the colostomy incision was removed by the thermo-cautery and hæmorrhage thus avoided.

Subsequently the patient made a slow but good recovery from the resection, and except for the evident lack of fluid in his tissues, from the cæcostomy still open, he appeared fairly well.

Third Operation—15 1 10. *Mr Morison.*—An incision was made round the margins of the cæcostomy opening and deepened down to the bowel. The skin edge and margin of the opening in the cæcum were invaginated into the bowel by interrupted catgut sutures, covered by lemberts. The wound in the belly wall was then closed as accurately as possible in layers, these being somewhat difficult to separate.

27/1/10.—The cæcostomy had not leaked since its closure and was then healed. The inguinal colostomy was acting well. The middle-line incision, which had gaped, was now clean, though there was still a fair area to granulate over. The patient's general condition had improved very much, and he left hospital feeling much better.

He returned at the end of September looking and feeling remarkably well.

Case .—*Malignant Growth of Sigmoid Excision—Permanent Colostomy.*—J. M., age 64, widow. Admitted 8/7/10. Discharged 2/8/10.

Patient gave a 4 months' history of illness, being quite well previous to this. Her trouble began with the onset of pains, which resembled those of labour, only referred to the "back body"; they were intermittent, and the attacks lasted about 5 minutes. As the pain passed off the patient felt inclined to micturate and passed a good deal of flatus. Medicine gave her some relief.

Six weeks before admission the pain became more severe in character and was associated with vomiting, which gave some relief; rumblings were now much more evident with the pain.

During the last 3 weeks the pain had been aggravated still more and the rumblings more marked. The bowels were constipated. Enemas gave some relief to the symptoms, but these had continued more or less the whole time.

The only urinary trouble was the desire to micturate when the pain was present.

On admission patient looked older than her years, and had a drawn and pinched expression. Tongue moist, slight fur; temperature, 99°; pulse, 100, good volume.

Local.—Abdomen slightly distended; the enlarged transverse and descending colon could be outlined on the belly wall. There was some tenderness in the left iliac fossa, and distinct peristalsis could be elicited in the colon by massaging it.

Per rectum and per vaginam a hard nodular growth could be felt high in the bowel. No history of inability to pass flatus.

Diagnosis—Cancer of lower end of sigmoid.

Operation—12/7/10. *Mr Morison.*—Patient took the anæsthetic badly at first, but improved later. An incision was made in the left iliac fossa, parallel to Poupart's ligament, and extending from the anterior superior spine to within 1½ ins. of the symphysis pubis.

On opening the abdomen and exploring the sigmoid a constricting growth was found, quite removable. The affected portion of bowel was drawn out of the wound and divided between clamps by the thermo-cautery, 2½ ins. on either side of the growth. It was excised with a considerable portion of mesentery. No enlarged glands were discovered. The distal end was completely closed by a continuous catgut suture, covered in by lemberts. The proximal end was drawn out of the wound and a Paul's tube inserted and fixed with strong silk. The descending colon was fixed to the belly wall by a continuous catgut suture on the inner side, and the rest of the wound was closed in layers, the deeper with catgut, the skin with silkworm.

Pathology.—The growth, as shown by microscopic examination, was "a carcinoma of the constricting variety," causing an annular stricture of the gut, so that the lumen would only allow of the passage of a penholder. The growth extended for 1 in. on either side of the constriction. No glands were discovered.

Subsequent.—Later in the day the patient complained of pain in the abdomen; the contents of the bowel were draining well by the Paul's tube; pulse thin and thready.

13 7 10.—Patient easier—still weak, stimulants ordered. Somewhat chesty.

17 7 10.—Paul's tube separated; patient much better; oil given; colon to be washed out.

2 8 10.—Wound quite healed; patient well; left hospital.

CASE .—*Cancer of Pelvic Colon.*—A female, aged 75, was admitted to the Royal Victoria Infirmary, 20 8 10.

History.—Until 7 days before admission she was quite well. Whilst doing her work she was suddenly seized with a stabbing pain in her abdomen, and vomited. Then her abdomen began to swell, and she had painful rumblings, loud enough to be heard in the next room. The bowels had latterly been very constipated, and she had had no motion since the attack commenced, though some flatus had been passed.

A year previously she had an attack of diarrhœa and passed slime but no blood.

She had never been jaundiced or had windy spasms.

On admission the patient looked fairly well; her tongue was moist and brown; pulse, 72; temperature, normal.

The abdomen was generally distended, and the dilated tense

cæcum could be distinctly felt. During examination increased peristalsis was evident. Flatus passed after admission.

Per Rectum.—Marked ballooning of rectum, but no growth. Higher up a hard swelling could be felt through the rectal wall.

Diagnosis.—Cancer of pelvic colon.

Operation—25/6/10.—The abdomen was opened through an oblique incision in the left iliac fossa. A growth freely movable was found in the sigmoid flexure, 6 ins. from its termination, in the rectum. No secondary deposits in the liver or elsewhere could be found. Above the growth the intestine was enormously distended. A loop of sigmoid above the growth was drawn out and opened and a Paul's tube was tied in.

The patient was entirely relieved by this operation, and slowly but steadily improved.

Second Operation—2/8/10.—Eight inches of the bowel, including the growth, the colostomy opening, and the corresponding mesentery, were excised, and end-to-end anastomosis was performed by the invagination method, *i.e.* by a tube tied in the upper segment and drawn down through the lower. The iliac abdominal wound was closed in 3 layers.

For the first 3 or 4 days the patient's progress was satisfactory, then her mind began to wander; finally she became restless and maniacal, and died 20/8/10 (18 days after the second operation).

Post-mortem examination, limited to the abdomen, showed that the abdominal wound was septic and infiltrated with pus. The omentum was firmly fixed over the operation area, and everything was so matted together that it was impossible to make any observation worth recording of the condition of the anastomosis. There were no glandular enlargements or secondary deposits.

Case .—*Carcinoma of Lower Pelvic Colon—Resection—Anastomosis.*—Female, aged 42, admitted to private hospital, 27/6/10.

History.—For 4 years the patient had trouble with her bowels. Previous to this she was quite well. During the first $3\frac{1}{2}$ years she had had attacks of pain with constipation, but in the intervals enjoyed excellent health. During the last 6 months she had frequent cramping pain in the abdomen, with wind rumblings, bad constipation alternating with diarrhœa, blood and slime in the stools, and had lost weight.

On admission she was a thin sallow woman, with tongue clean, temperature and pulse normal, and no sign of disease except in her

abdomen. The abdomen was quite lax. In the left iliac fossa a well-defined hard sausage-shaped tumour, its long axis in the direction of the bowel, could be felt rolling under the examining fingers.

Diagnosis.—Carcinoma of the pelvic colon, causing chronic intestinal obstruction.

Operation—29/6/10.—A long oblique incision, extending from the anterior part of the left flank to the middle line in front, dividing the left rectus abdominis muscle and deep epigastric artery, gave an excellent exposure. The growth was found to be of the constricting type of carcinoma, and was situated at the lower part of the sigmoid flexure. Some enlarged glands were felt in the mesentery close to the bowel. There were no signs of secondary growth elsewhere in the abdomen. After mobilisation the lower half of the sigmoid with its mesentery was excised, and an end-to-end junction of the upper part with the rectum was made by the invagination method. The rectum and the mesentery of the pelvic colon were then sutured to the fundus of the uterus to give additional support to the union. The abdominal wound was closed in layers.

Pathologist's Report.—The tumour is a columnar-celled cancer, definitely infiltrating the submucous and muscular coats.

Nine days after operation the bowels were moved normally, and the tube was evacuated. Recovered.

CASE .—*Carcinoma of the Pelvic Colon forming a large Pelvic Tumour.*—Female, aged 45, married, was admitted 1/10/10.

History.—Five months ago her illness began with an attack of acute intestinal obstruction, which was followed and relieved by diarrhœa. Since then she had not been well and had constant diarrhœa, sometines blood in the motions, and painful micturition. Menstruation, previously regular, had ceased for 2 months.

On admission she looked ill, pale and sallow; tongue, moist and clean; pulse, 96; temperature, 99.6°. Her urine contained albumen.

The abdomen was distended below by a large firm mass with a smooth surface and well-defined rounded edges, fixed in the pelvis below, but with a limited side-to-side movement above. There were no signs of free fluid. Per vaginam the uterine cervix was pointing backwards and was somewhat fixed. The left fornix was pushed down and Douglas's pouch was occupied by a hard tumour pushing the uterus backwards and to the right. The impression

conveyed to the fingers was that the lower segment of the uterus was fixed in cement.

Diagnosis.—Large left malignant ovarian cyst filling the upper part of the pelvis, with infiltration and inflammation of the lower portion.

Operation—18/10/10.—The abdomen was opened in the middle line below the umbilicus, and the patient was placed in the Trendelenburg posture. Coils of small intestine were adherent over the top of the tumour, and in separating these and drawing up the mass a coil of infiltrated small intestine was torn. The diseased portion (4 ins.) was resected and an end-to-end anastomosis made with an inner through-and-through continuous catgut suture and an outer row of interrupted silk lembert sutures. A further attempt to remove the tumour showed that it consisted of an inseparable conglomerate mass, consisting of ovaries, tubes, broad ligament, uterus and intestines, and irremovable. Before closing the abdomen an enema was administered and some of it escaped into the pelvis, showing that the large bowel had been opened. The opening was small, low down, and inaccessible. Some iodoform paste was smeared over the position indicated by the escape of fluid, the omentum was tucked down over the opening and a drainage tube extending into the pelvis was left in the lower part of the wound, and a large indiarubber tube was left in the rectum and sutured to the anus. Four days later there was a fæcal discharge from the abdominal wound. Inguinal colostomy was advised, but refused, and the patient went home. Recovered. At home the wound and fæcal fistula closed for a time, then it broke down again, and the patient died 6 months after the operation.

Case .—*Malignant Growth in Sigmoid Flexure of Colon—Vesico-Colic Fistula—Colostomy—Death.*—T. O., age 53. Admitted 11/6/10. Died 20/6/10.

Patient admitted with a history of illness of 9 months' duration. He had complained of pain in both iliac fossæ, painful wind rumblings and frequency of defæcation, his bowels being moved about 6 or 8 times daily. The motions were unformed and offensive, and latterly he had passed white slimy material from the rectum. During the last three weeks he had been confined to bed, and had had 4 rigors. Three days before admission he began to pass flatus per urethram, and had increased frequency of micturition, with pain along the urethra during the act. The urine was very thick and offensive.

During the last 3 days also the motions had been darker in colour, and there had been some attempt at formation. He had lost 1 or 2 stones in weight during the last few weeks. He had never been abroad. He had previously been treated as a case of mucous colitis.

On admission patient was feeble and emaciated ; tongue, moist and furred ; teeth, false ; pulse, 128, regular, small volume, low tension ; temperature, 99.5° F.

Local.—No abdominal tenderness. He held himself so rigid that palpation was difficult. In the upper part of the left iliac fossa a mass could be felt on the posterior abdominal wall, which was thought to be large bowel. *Per rectum* there was ballooning, and on the anterior rectal wall above the prostrate an indefinite mass could be felt, suggestive of an inflammatory condition. Fæcal matter was present in the urine.

14/6/10.—Patient was passing a great deal of flatus per urethram, which caused him intense agony, while particles of fæces continued to be present in the urine.

Diagnosis.—Sigmoido-vesical fistula due to abscess in connection with a sigmoid sacculus.

Operation—15/6/10. *Mr Morison.*—Median incision, the umbilicus being excised. Patient took the anæsthetic very badly, and his rigid belly wall gave great trouble to the operator. The appendix was pathological ; its tip was bulbous and there were numerous adhesions around it. Appendicectomy was performed, the stump ligated, carbolised, and invaginated. In the neighbourhood of the division of the left common iliac artery a hard mass was found, which could not be definitely stated to be growth or inflammatory. Numerous coils of rather collapsed small gut were adherent to it. It was considered injudicious to separate these, and so the following procedure was adopted :—The sigmoid was divided above the mass, both upper and lower ends being closed ; the upper end was brought out through a separate oblique incision in the left iliac fossa, the lumen being left obliterated. The median incision was closed in deep layers of catgut, the skin being sutured with silkworm gut ; the bladder was thoroughly irrigated and soon washed clean. Paul's tube was fixed into the proximal gut. The bladder was subsequently irrigated twice daily.

20/6/10—(5 days after operation).—The patient was never fit after operation, and died in a condition resembling uræmia. Very little fæcal matter had passed through the Paul's tube.

Post-mortem.—A mass of porridge-like fæces were found in the cæcum and ascending colon. There was a malignant growth of the sigmoid and a fistula between the intestine and upper part of the bladder, to which area many coils of small intestine and also omentum were adherent. The kidneys appeared to be normal. There was no peritonitis, no evident sepsis, and no discoverable surgical cause of death.

Case .—*Multiple Rectal Polypi—Colostomy.*—J. R., age 13, schoolboy. Admitted 11/4/10. Discharged 19/5/10.

In August 1808 Mr Richardson removed a polypus from the rectum, after which he was well for some time. However, his old symptoms recurred, and might be shortly stated as follows :—He had *no pain* with defæcation. He sometimes had a profuse *discharge of bright red blood* on having his bowels moved, and this recurred from time to time. He had sometimes to be carried from the lavatory, quite collapsed from loss of blood. His *motions* were well formed and natural, except when the presence of blood altered their appearance. *Something frequently came down* after defæcation, and this had to be replaced.

On admission his general condition was good ; temperature and pulse, normal ; chest, clear.

Local—Per Rectum.—Numerous sessile papillomata could be felt situated all over the mucous membrane and varying very considerably in size ; they extended as high as the finger could reach.

Sigmoidoscopy—14/4/10. *Mr Hamilton Drummond.*—Numerous sessile papillomata could be seen, varying in size from a pea to a cherry, extending the whole length of the rectum, with very vascular tops, presenting a bright red appearance ; none were demonstrable in the pelvic colon, and so apparently they were limited to the rectum.

Operation—19/4/10. *Mr Morison.*—Left inguinal colostomy, first stage, performed. Loop of sigmoid pulled out and fixed with a clip through its mesentery ; no sutures introduced and the bowel not opened.

20/4/10.—Bowel opened longitudinally ; clip removed. Both colon and rectum washed out daily with saline.

19/5/10.—Bowels were being opened twice daily through the colostomy ; occasionally a little fæcal matter escaped per anus with the lavage. The colostomy opening to be left for 12 months, to

prevent the irritation of the fæces passing over the papillomata with the hope of cure.

Readmitted—19 5 11.—Since his operation his general condition had been greatly improved. He had had no hæmorrhage from the rectum, and had been able to play football. The bowels were opened by the colostomy about twice daily.

On admission he looked physically fit. There was some prolapse of mucous membrane through the colostomy opening. *Per rectum*, on digital examination, no polypi could be made out, but little definite could be ascertained on account of some putty-like fæces which was present. Repeated enemata failed to clear this away, and it had ultimately to be removed by digital means. Sigmoidoscopy now showed an entire absence of polypi, but there were one or two small areas, like raspberry nodules, in the region where the polypi had been. Whether these were the last signs of polypi or were dependent on the presence of fæces for their existence could not be settled. The sigmoidoscope was introduced for 8 ins. It could not be inserted from the colostomy opening on account of the smallness of its size. On digital examination a few very small hard nodules, like split peas, could be felt, apparently in the rectal wall. Whether these were remains of old polypi was not certain, but it was decided to postpone closure of the colostomy a little longer, in order that they might be given a chance of disappearing.

Case .—*Colitis with Ulceration and Polypi—Cæcostomy—Death.*—H. B., age 27, married. Admitted 21 1 10. Died 28 1 10.

Patient admitted complaining of the passage of blood and slime per rectum, accompanied by pain.

Her illness began 5 years ago, after the birth of a child.

Pain.—Varied from time to time, at intervals being very bad. It had been worse during the last 2 weeks, spasmodic and shooting in character and accompanied with the desire to have the bowels moved. On attempting to defæcate there was frequently only a *discharge* of blood and mucus, while with every motion these were present, but not in any great amount at one time. She had been confined to bed and was very ill for the last month.

Stool.—She complained of constipation as a rule, not considering the discharge a motion ; with the latter there was really diarrhœa. She had had to be careful with her diet for the last year, chiefly avoiding meats, pastry, and green vegetables ; eggs and milk suited her best.

Anything coming down?—From the onset, with straining, the backbody came down, but this was considered to be nothing more than piles.

On admission she was a thin, anæmic, very ill-looking woman. Breath, very foul; temperature, 99.6°; pulse, 100.

Local.—The abdomen looked normal. There was tenderness over the cæcum, and a sausage-shaped tumour could be felt in this situation, like the bowel. Nothing else was to be felt.

Per Rectum.—Small hard papules could be felt scattered all over the rectal mucous membrane, varying in size from a pin-head to a pea.

Sigmoidoscopy.—Red raised areas could be seen on the mucous membrane surrounded by superficial ulceration; these areas were the papulæ felt. The condition extended right up into the pelvic colon, and only the lower part of the latter could be examined from the presence of blood and mucus, apparently coming down from above. The whole bowel seemed much thickened.

Diagnosis.—Multiple sessile polypi, with ulceration. Extensive ulceration, leaving areas of mucous membrane; cause unknown.

Operation—25/1/10. *Mr Morison.*—Cæcostomy performed in the usual way and Paul's tube inserted. The cæcum appeared normal.

28/1/10.—Patient gradually sank, making no improvement, and died.

Specimen obtained post-mortem showed the whole rectum, pelvic, descending, and half the transverse colon to be the seat of disease. The lower in the bowels the worse the disease appeared, and the rectum and pelvic colon were studded with what appeared to be multiple polypi, and here the disease was worst. Higher up the bowel the appearances were more those of multiple small ulcers.

Microscopic Report.—The whole condition was one of chronic inflammation with ulceration, and certainly not due to either new growth or tubercle.

Case .—*Sigmoido-Vesical Fistula due to Sacculated Sigmoid.*—Female, aged 61, married, admitted to private hospital, 31/8/10.

History.—This was indefinite. All that could be elicited from the patient and her husband was that she had been constipated, and 7 or 8 years ago noticed flakes in her urine, but only at times. Sometimes increased frequency of micturition occurred and the urine was very offensive in smell and dark coloured. Occasionally

clots of blood were passed, causing great pain. The condition had apparently caused very small trouble.

On admission the patient was a very stout, healthy, though somewhat pale-looking woman. Her doctor had sent along with her a sample of her urine, which contained gross fæcal matter.

In the hypogastrium to the right of the middle line an ill-defined tender mass was discovered on palpation. Per vaginam the uterus was fixed posteriorly by hard exudate in the pouch of Douglas. Except for the fæcal contents there was little found in the urine, and the amount of urea passed in 24 hours was normal.

Cystoscopic examination (Mr Willan) showed a circular opening large enough to admit a lead pencil behind the left ureteric orifice at the junction of the left lateral and posterior walls on the base of the bladder. The rest of the bladder was normal. It was difficult to keep the bladder distended for cystoscopic examination as the fluid escaped into the rectum and ran out of the anus. Sigmoidoscopic examination revealed nothing.

Diagnosis.—Sacculitis of sigmoid with ulceration, adhesion to bladder and fistula.

Operation—10 8/10.—The abdomen was opened in the middle line below the umbilicus. There was considerable matting of intestinal coils, which were firmly adherent in the pelvis. It was finally determined that a loop of the ileum and a portion of the sigmoid flexure low down were firmly adherent to the bladder wall. In separating these adhesions the fat and brittle mesentery of the small intestine was separated for a considerable distance from the gut, and the adherent sigmoid was torn completely across.

The lacerated portion of sigmoid was resected and the upper end was invaginated into the lower by a tube fixed into the upper and passed from above through the rectum and anus. Nine inches of the damaged ileum close to the ileo-cæcal valve were next resected, the cut ends were closed, and a lateral anastomosis was made with a Murphy button (any other means of making the anastomosis would have been exceedingly difficult, as the intestine was so small and was buried in a very fat mesentery). The hole in the bladder was then carefully closed by interrupted silk sutures. (On account of the depth of the pelvis and the fat patient this was a very difficult proceeding.) The pelvis was drained through a hole made at the bottom of Douglas's pouch into the vagina, a catheter was fixed into the bladder, and the abdomen was closed with 3 tiers of suture.

Towards the end of the operation 2 pints of saline were given intravenously.

The operation occupied 2½ hours, and was the most arduous I have undertaken or seen.

Recovery was delayed by suppuration and the formation of a fæcal fistula.

Discharged 20/11/10. Well.

Case .—*Gastrocolic Fistula.*—Male, aged 39, was admitted to private hospital 23/11/10.

History.—The patient was quite well till 5 months ago, when he began to have a feeling of fulness in the stomach and to lose flesh.

About the middle of September (2 months ago) he felt a "boiling" sensation in the stomach and made himself sick to obtain relief. He vomited about 2 pints of foul material which smelt and looked like fæces.

Ten days ago he experienced the same symptoms and vomited as before.

The day before admission his doctor had washed a quantity of foul fæcal-smelling material out of his stomach.

There had never been acute pain in his stomach, but there had often been a dull ache.

Lately he noticed that his bowels had been moved 3 times daily, in fact after every meal, and he had been surprised at the size of his evacuations. He had never noticed food in the motions. His appetite had been, and still was, enormous.

On admission the patient was a pale, thin man, full of energy and hope; tongue, clean; breath, foul and fæcal; temperature and pulse, normal.

On examination of the abdomen there was marked epigastric rigidity, especially on the left side, and local tenderness over the upper left rectus. No mass could be felt. Nothing abnormal elsewhere was discovered. A bismuth meal was given to him, and its progress was watched by Mr Hamilton Drummond by X-ray through a screen, with the patient standing up. First the stomach was seen to be distended by the meal, then almost immediately it was seen to escape into the colon to the right of the splenic flexure. Colon peristalsis was quite plainly visible. Within half an hour on his return to the hospital an enema was administered, and the greater portion of the bismuth porridge was passed with it.

Diagnosis.—Gastrocolic fistula due to a malignant growth connecting the stomach and transverse colon.

Operation—31/11/10.—The abdomen was opened in the middle line above the umbilicus. There was an extensive and inoperable malignant growth of the stomach, chiefly on its posterior wall, to which the transverse colon was firmly attached. Recovery.

Case .—*Ulceration and Stricture (Malignant?) of Colon—Recto-Vaginal Fistula.*—Female, single, aged 36, was admitted to private hospital, 10/10/10.

History.—All her life she had been much troubled by constipation. Sixteen years ago she contracted "dysentery" after a chill caught whilst sea-bathing. Since this attack her bowels were so loose that if she wished to go anywhere she had to take a sedative, the effect of which lasted for 6 hours. In June 1910 (4 months ago) she began to have spasms of pain across the abdomen, which lasted for 1 or 2 hours. Two months later these became very severe, were accompanied by vomiting, and lasted for about 18 hours. Six weeks ago she had intense pain, diffused over the lower abdomen and extending into the left groin and the vagina. She was also troubled with hæmorrhoids. After 10 days of suffering she felt something burst and there was a free discharge of blood and pus from the vagina. The pain was relieved, but a profuse discharge continued. Fæces then began to appear in the discharge, and finally for a week before her admission all the fæces escaped through the vagina, nothing coming from the anus.

She had lost weight and had been unable to sleep without an opiate on account of the pain and discharge. Menstruation ceased 3 months ago.

On admission her general condition was poor. Temperature, 100°; pulse, 100.

The abdomen was not distended, and palpation only discovered some tenderness in the left iliac fossa. Under an anæsthetic the rectum was found to be extensively ulcerated, extending from the anus as far as could be reached. A large hole admitting the tip of the index finger communicated with the vagina just above the anus. The vaginal wall presented a large ulcer, with hard undermined edges extending upwards on the left and undermining the labium minus.

Diagnosis.—Ulcerative colitis, with stricture and partial obstruction.

Operation—12/10/10.—The abdomen was first opened by an oblique incision in the left inguinal region. The sigmoid flexure was bound down by adhesions thickened, contracted, red, and covered by excrescences like the comb of a cock. (One of these was removed for pathological report.) The remainder of the colon was next explored, and it was found to be in a similar condition though less in degree throughout. A second oblique incision was made in the right iliac fossa over the cæcum. It was seen to be reddened and thickened, and felt hard and œdematous. The veriform appendix was much thickened, distended, and obviously diseased. It was excised. The cæcum was then fixed to the abdominal wall (first stage of cæcostomy). Pathologist (Professor Stuart M'Donald) reported :—

1. Of the anal ulcer.—" This appears to be a simple ulcer. There is nothing to suggest malignant disease or any of the infective granulomata."

2. Of the appendix.—" It shows chronic inflammatory changes in the wall, with lymphocytic infiltration of muscular and epithelial coats."

3. Peritoneal nodule.—" This shows a group of tubular glands. The tubules are lined by tall columnar cells, among which some goblet cells are present. The stroma is cellular and vascular. If this nodule came from the peritoneal coat of the intestine I cannot explain the appearances, except on the hypothesis of a secondary deposit of columnar-celled cancer."

Four days later the cæcum was opened to establish an artificial anus. Secondary hæmorrhage occurred several times from the cæcostomy wound, but ultimately ceased. The improvement in this patient's condition was remarkable, though it was very soon impossible to wash the colon from the cæcostomy wound to the anus or *vice versâ*. She returned home, 9/12/10. Recovered, to return in 1 year for consideration of the question of colectomy.

Case .—*Ruptured Duodenal Ulcer and General Peritonitis—A Diagnosis of Intestinal Obstruction.*—A male, aged 39, was admitted to the Royal Victoria Infirmary, 7/7/10.

The patient was sent into the infirmary as an emergency case, suffering from " retention of urine."

History.—His illness began a month ago with loss of appetite, and he had lived on fluid foods. A week later he felt a gnawing

pain in the stomach while he was at work. He went home, had tea, and afterwards vomited.

Since then he had had pain in the abdomen more or less constantly, varying in severity, at times bad enough to make him stop work. He vomited about once every day, a fairly large quantity, sometimes bile-stained; it relieved the pain for a time. In addition to the vomiting he was troubled a good deal with acid eructations. The relation of pain to his meals was indefinite, as he was only taking fluids. During the past week he had passed little urine, and had considerable pain with micturition. Three days before admission he had an acute exacerbation of pain; it remained worse than before. Two days before admission he developed complete retention, for which a catheter was passed. During the last 2 days he had passed no flatus or fæces, and had eructated much gas.

He said that he had had many similar attacks to the last, but with each successive one his symptoms had been worse.

On admission he walked in, but with difficulty, and appeared to be very weak. He was pale and anxious-looking, much emaciated; temperature, 100° F.; pulse, 110. The abdomen was distended and tense. No peristalsis was seen, no rumblings heard, and no mass felt. On percussion there was considerable dulness over the pubes, but the remainder of the abdomen was tympanitic. After passing urine, which he did unaided, the suprapubic dulness did not disappear, and as it was doubtful whether the catheter used had entered the bladder, because there were several false passages, an aspirator needle was introduced into the dull area and about 1 dr. of fluid with a urinous smell withdrawn. Subsequently the patient passed his urine naturally, but neither flatus nor fæces.

9.7.10.—Patient passed a little flatus for the first time since admission. The distension of his abdomen was less. Nothing could be felt except that both recti muscles were rigid. His general condition was very feeble, and he was pained and sick. A pint of black watery fluid without fæcal odour was washed out of his stomach.

Diagnosis.—Intestinal obstruction, with peritonitis.

Operation—9.7.10.—On opening the abdomen the pelvis was found to be filled with purulent dirty-looking fluid. The whole of the small intestines were red and distended, and covered with purulent lymph. The cause of this was found to be a ruptured duodenal ulcer, about $\frac{1}{4}$ in. in diameter, immediately beyond the pylorous. The duodenal opening was closed by a double layer of

sutures, the abdomen thoroughly flushed out with normal saline, a drainage tube left in above the pubes, and the abdominal wall closed in layers. During the operation 2 pints of normal saline were introduced into the circulation through the median basilic vein.

For the first 3 days after operation the patient made such satisfactory progress that his recovery was anticipated. On the evening of the third day (12/7/10) he became restless, then delirious, and his pulse and temperature, which had dropped to normal, rose to 140 and 100.4° F. respectively. A submaxillary gland next swelled, and a week later an abscess was opened under local anæsthetic. Then his breathing became embarrassed, and he died on the evening of 21/7/10 (12 days after the operation) of pneumonia.

Post-mortem examination showed that his death was due to septic broncho-pneumonia. His abdominal condition, except that there were traces of lymph here and there on the intestine, presented nothing abnormal.

CASE .—*Appendix Abscess which had leaked into the Cæcum, leaving a Hard Mass simulating Ileo-Cæcal Cancer.*—J. M., age 63, dealer. Admitted 18/2/10. Discharged 4/3/10.

Three weeks previous to admission patient had a single attack of pain in the upper abdomen after a heavy meal, and was blown up with wind. He vomited and obtained relief, and remained well for a week. At the end of this time he felt poorly all day, and by 2 A.M. he had developed a very severe pain in the abdomen, diffuse and continuous ; he did not vomit. The following day the pain settled in the right iliac fossa, and he was subsequently tender to examination in this region. His doctor told him something had formed. The pain gradually decreased in severity, and at the time of his admission he had not any, and was only complaining of a feeling of weight in the right lower abdomen. He had had no trouble with micturition. His bowels had been somewhat constipated, but had been moved by enema 2 days after the acute attack. He had had no diarrhœa, and had noticed no blood in the stools. In addition, there had been no rumblings in his abdomen, and he vouched that he was always able to pass flatus. There was no history of any previous attack. He was rapidly losing weight.

On admission patient was in fair condition ; temperature and pulse normal ; chest clear.

Local.—Abdomen quite lax ; small mass felt in the right iliac fossa, only slightly tender on pressure, hard, nodular, and somewhat

movable. The resonance in this region varied, and a thrill, obtained by flicking the belly wall, was put down to the laxity of the parietes. There was no liver enlargement, and no other mass was felt in the abdomen, nor could glands be felt in the left posterior triangle of the neck. Per rectum, no mass felt bimanually; prostate considerably enlarged.

Diagnosis.—From the age of the patient and the nature of the mass felt it was decided to be a case of ileo-cæcal carcinoma, the alternative diagnosis being that of old appendix abscess.

Operation—22/2/10. *Mr Morison.*—Usual appendix incision; belly muscles divided. They showed marked œdema. On opening the abdomen there were present some omental adhesions to the anterior belly wall. The appendix was found wrapped round by, and buried in, inflamed omentum. On separating this the remains of an old abscess between the appendix, omentum, and mesentery of the ileum were exposed. The cæcal wall was thickened and œdematous, and showed a small hole, which, however, did not communicate with the lumen of the gut, but which was considered to be the seat of discharge of the abscess into the cæcum. The appendix exhibited a very bulbous tip, and the remainder of it, which was very long, was shelled out of a very thick œdematous peritoneal coat, and removed in the usual way. The thickened coat and omentum were sutured over the stump, and the abdominal wound closed in layers without drainage.

4/3 10.—Uninterrupted recovery; patient left hospital healed and quite well.

2. ENLARGED PROSTATE ASSOCIATED WITH VESICAL TUMOUR. NOTES OF SIX CASES

By David Wallace, C.M.G., F.R.C.S.,
Surgeon, Royal Infirmary, Edinburgh

As operative experience increases in cases of enlarged prostate, liability to error in diagnosis based on symptoms alone is necessarily more recognised. The fallacy of fibrous stenosis at the neck of the bladder as a cause of prostatism is now admitted, and the need for care in differentiating simple enlargement from malignant disease of the prostate is well known. Within the last two years I have seen six cases of enlargement of the prostate associated with the presence of a vesical neoplasm, in each of which

the symptoms were explicable from the enlargement of the prostate without any suggestion of the presence of a tumour, and it is to this possible association that I wish to direct attention. In all of these cases hæmaturia was a prominent symptom, but this did not necessarily indicate the presence of a tumour, as it is quite well known that profuse bleeding may be due to prostatic enlargement alone, this symptom of bleeding not infrequently being the first symptom seriously to direct a patient's thoughts to his condition, so that he consults his medical attendant. More especially is this the case when clots are passed and difficulty in micturition results, leading, it may be, to temporary retention. If a patient comes before us with a history of bleeding and he is well up in years, describes symptoms such as we are familiar with in prostatic cases, and we find on examination *per rectum* that the prostate is enlarged, it seems natural to take the simple explanation and diagnose hæmaturia a result of prostatic enlargement. In such a case as a routine we examine the kidneys by palpation, but unless we have definite evidence of something wrong, we are almost justified in believing that the prostate is the cause. We do not consider that it is necessarily a malign prostate, because we are more likely to have profuse bleeding in a simple case than in neoplasm of the prostate. At the present time prostatectomy is so often recommended, and its results are so beneficial in the majority of cases, that we are most likely, having made the diagnosis of enlarged prostate, to advise operation and give a very favourable prognosis. If, however, at the operation a vesical tumour is found, it must be very disconcerting to the surgeon and most disappointing to the patient. On the other hand, if the symptoms are not urgent and the bleeding not profuse, operation may be delayed, palliative measures adopted, and valuable time is lost. As an example of this I quote the following case :—

G. D., æt. 70, in April 1909 complained that he had to rise three or four times at night, and that for three weeks he had seen some blood in his urine. It came at the beginning, and the urine towards the end was clear. He had no pain in the lumbar regions and the kidneys were not palpable. *Per rectum* the prostate was not much enlarged, and there was no induration. He stated that the first bleeding was seven years previous to the present attack and lasted eight days. Since then bleeding had occurred three or four times and lasted for a day or two. I advised no operation, and wrote to his doctor that I thought the condition was prostatic,

but that if the bleeding continued he should be examined cystoscopically. In October 1910, that is about eighteen months later, he came to see me again. The bleeding had ceased for nearly a year, and then recurred. The frequency of micturition was as before, but he now stated that he had lost weight. Examination *per rectum* revealed practically no change. I examined him cystoscopically and detected a villous tumour just above the left ureteral orifice. I later removed this tumour and the patient made an excellent recovery.

One naturally asks the question in regard to the above case, Should an earlier diagnosis have been made of vesical growth? and I am satisfied that a similar history of bleeding in a man of about 50 would have at once suggested a vesical neoplasm; but in a man of 70 the symptoms were rather those of enlarged prostate, and had he had greater micturitory trouble I would probably at the first have advised prostatectomy without the use of the cystoscope as an aid to diagnosis.

Take another case—Mr A., æt. 69. I need not detail the symptoms further than to say he had frequency of micturition, and at the time I saw him, hæmaturia, the former of some years' duration, the latter, of much more recent origin, three months. The prostate was enlarged, firm, and rather fixed, so much so that I feared it might be malign, and it was certainly not a satisfactory one for removal. Again I attributed the symptoms to prostatic enlargement, with the *caveat* that the prostate might be malignant. I advised no operation, but said that if the symptoms got worse, or the bleeding increased, a cystoscopic examination should be done with a view to immediate operation if that proved feasible. In six months, his symptoms being worse and the bleeding more continuous and profuse, I made a cystoscopic examination, which revealed a large sessile papilloma, involving the bladder-wall so widely that removal was out of the question. I performed a suprapubic cystostomy, and he wore a tube for some months with much comfort.

In this case, again, if the patient had been a younger man I think the diagnosis of vesical tumour would have been arrived at from the symptoms, but at the patient's age these were quite explicable on the ground of an enlarged prostate, and a provisional diagnosis was made on that basis.

The other four patients were all over 65 years of age, and the symptoms in each were those of hæmaturia associated with

prostatic enlargement rather than caused by bladder tumours. In only one was there pain accompanying the bleeding, and in his case another surgeon had sounded him for stone three years previously, and failing to detect a stone pronounced the condition to be due to enlarged prostate. The bleeding ceased for months, but the frequency of micturition continued to give trouble, both during the day and at night. I saw him when the bleeding had continued for several months, and concluded that the condition was due to a tumour. This I verified by cystoscopy, when I detected a large villous growth. A palliative suprapubic cystostomy was performed, and the bleeding entirely ceased.

The conclusion of the matter may be summed up as follows :—

Hæmaturia is a symptom of prostatic enlargement and also of vesical tumour, and in a man with enlarged prostate it is often not possible to say it is due to the one rather than to the other. Short of operation there is only one means of diagnosis, and that is by cystoscopy.

When we have much bleeding, or when bleeding comes on without any aggravation of other symptoms in a case of enlarged prostate, no instrumentation having been used, it should be borne in mind that a neoplasm may be the cause. In such circumstances it is judicious to make a cystoscopic examination prior to opening the bladder. In some cases it may not be possible to pass the cystoscope, and then we must operate, but in such a guarded prognosis should invariably be given.

Meeting IX.—July 5, 1911

DR BYROM BRAMWELL, *President, in the Chair*

I. ELECTION OF MEMBER

George Fyfe, M.D., 24 Broughton Place, was elected an Ordinary Member of the Society.

II. EXHIBITION OF PATIENTS

1. *Dr Chalmers Watson* showed—

Case of MYASTHENIA GRAVIS.

Man, æt. 41, paraffin worker ; pains throughout the body and

general weakness, of six weeks' duration. Since the New Year patient has been getting gradually weaker and less fit for work. Clinical condition characteristic of myasthenia gravis.

2. *Dr Lovell Gulland* showed—

A case of LOCOMOTOR ATAXIA, treated with Salvarsan.

3. *Mr Charles W. Cathcart* showed—

GALL-STONES complicated with cyst in head of pancreas—removal of gall-stones—drainage of gall-bladder, common bile duct, and cyst.

Mrs H., aged 40, was admitted to ward 17, Royal Infirmary, on April 12th, 1911. About fifteen months previously, when three months pregnant, she took a chill and this was followed by severe pain in the upper part of the abdomen, with vomiting and jaundice. Other attacks followed with intermissions until the baby was born. After a month's interval the attacks returned and continued steadily. The last one only ceased ten days before her admission.

During these attacks she becomes jaundiced, and vomits several times a day, bringing up a bitter, greenish fluid, sometimes blackish material. She loses appetite, and as all food causes pain, she practically starves herself.

On examination, no swelling nor tenderness could be made out in the region of the gall-bladder, but on deep palpation a mass was felt in the epigastric region a little to the left of the middle line.

When admitted to hospital, *i.e.* during an interval between attacks, she had only slight abdominal pain, and no jaundice.

Operation, April 17th, 1911.—The abdomen was opened through the right rectus muscle, and the gall-bladder, pear-shaped, and enlarged, was found hidden under the border of the liver. It was drawn forward along with the liver, and opened. It contained three or four rounded gall-stones about the size of small marbles, lying in a bile-stained fluid. The wall of the gall-bladder was thickened, apparently from chronic inflammation, and at one part —adjacent to the liver—contained a cyst.

The common bile duct was opened but no stone could be felt in it. The probe, however, did not pass easily down it, probably on account of a swelling in the head of the pancreas. This swelling, when exposed by dissecting through the transverse meso-colon, was found to be a cyst about the size of a large walnut. It was

drawn forward to the abdominal wound and emptied of about half an ounce of slightly turbid glairy fluid. The cyst wall was then stitched to the peritoneal margin of the wound and a drainage tube placed in position. The gall-bladder was in like manner fixed and drained, and the rest of the anterior wound was closed.

A tube was fixed in the common bile duct and brought through a stab wound in the loin.

The wound healed well and the patient was discharged convalescent on May 22nd.

4. *Dr G. A. Gibson* showed—

(*a*) Patient, aged 41, complaining of uneasiness in chest and breathlessness on exertion. Vessels healthy; arterial pressure 92 diastolic, 133 systolic; pulse regular and equal; heart slightly enlarged; long late systolic thrill continued beyond second sound; harsh murmur through similar period; also separate aortic diastolic murmur; no shadow above that of heart (by skiagram) more than normal. Diagnosis: patent arterial duct with aortic disease.

(*b*) Patient, aged 44, admitted with purpura in fifth year of syphilitic infection. Fusiform dilatation of aorta discovered. Great reaction on use of salvarsan, followed by left hemiplegia. Arterial pressure throughout never exceeded 90 diastolic and 140 systolic.

III. Exhibition of Specimens

1. *Mr Charles W. Cathcart* exhibited—

Carcinoma of kidney invading inferior vena cava.

Hugh R., aged 55, was admitted to ward 18, Royal Infirmary, Edinburgh, for radical cure of hernia. He had no symptoms pointing to kidney trouble till on the morning fixed for operation he had hæmaturia. The operation was therefore postponed. He had a slight stricture. After the use of the cystoscope he developed a perineal abscess, with sloughing of the tissues, and external urethrotomy was performed. He improved for a time, but afterwards grew steadily weaker till he died, about 4 months after admission. He was anæmic, but had no more hæmaturia. For a week or two before death his feet and legs were œdematous, but the veins were not distended, and there was no suspicion of obstruction of the inferior

vena cava. Tuberculous disease of the left kidney was thought probable.

2. *Dr Eason* showed—

Lungs from boy aged 9. History of cough and emaciation.

Typical signs of right pneumothorax noted on admission to hospital. Death a few hours later. Right pleura was greatly distended, and corresponding lung collapsed. Rupture of a basal cavity into pleural sac. Tuberculous broncho-pneumonia of both lungs.

IV. Original Communication

A CASE OF LYMPHADENOMA TREATED WITH SALVARSAN

By G. Lovell Gulland, M.D., F.R.C.P.E., Physician to the Royal Infirmary and Royal Victoria Hospital, Edinburgh, and A. Goodall, M.D., F.R.C.P.E., Lecturer on Physiology, Surgeons' Hall, Edinburgh.

It is so rare to get even temporary benefit from any medication in cases of lymphadenoma when they are once fully established, that we have thought it worth while to put this case on record. The patient, J. L., was a bank clerk of 24 years of age. There was no point of importance in his family or previous history, except that three years before the onset his tonsils had been removed. In October 1909 two glands appeared low down on the left side of the neck, about the size of marbles. He saw a doctor in Glasgow at that time, who suspected lymphadenoma, and gave him iodine and arsenic internally, without much benefit. During the winter of 1909-10, the patient got through his work fairly well, but towards the end of it complained of weakness in the legs. The lumps had at first been soft, but gradually got harder, and slight swelling appeared in the left axilla. In April 1910, he began to have attacks of fever lasting a day or more, with a temperature rising to 101° or 102°, and he suffered from profuse perspiration at night, with considerable weakness. Von Pirquet's test at this time was negative. He had, by this time, gone to his home in Blairgowrie, and Dr Shaw sent him down to see one of us on June 11th, 1910. At that time he looked well, the lungs, urine, abdominal organs were all normal, the heart was slightly dilated, the blood

count was—reds 6,400,000, hæmoglobin 95 per cent., colour index .74, whites 13,300, with the proportions of polymorphs 75, lymphocytes 24, and eosinophils 1. The only glands which could be felt were in the left side of the neck, immediately above the clavicle, and another mass also beneath and behind the sterno-mastoid. The glands at the angle of the jaw were not enlarged. There was another mass in the left axilla. At that time the glands were rather soft, and there was a certain amount of œdema and swelling about them. We decided then to put him on arsenic subcutaneously, as he had been taking it by the mouth in 6 or 7 minim doses all winter. We also gave him tuberculin T.R., beginning with $\frac{1}{20,000}$th of a milligram, which was to be increased steadily. Dr Shaw excised one of the smaller glands, and sent it down to us for examination. It showed nothing very definite. Thereafter, the patient stayed at home in Blairgowrie all summer. The glands became smaller, and he had no rise of temperature for a long time. The arsenic was given hypodermically every second day, up to doses of 20 minims, which was reached about the 20th of August 1910. Thereafter he began to lose appetite, and had a febrile attack, and had four of these between that time and the middle of October. The course at this time was, the temperature gradually rose for three or four days until it reached 103°, and then gradually fell in about three days to the normal. These attacks had no relation whatever to the tuberculin injections. The enlarged glands had receded up to the 20th of August, but with each of these febrile attacks they became softer and more swollen, and more glands at the margin were involved. He was seen here again in the beginning of December, by which time the febrile attacks had settled down into greater regularity. They lasted seven or eight days, he was free for ten days, and then another attack started. The glands in the neck had by this time become pretty hard, those in the axilla were perhaps a little increased in size and softer, and there was enough cogwheel breathing at the left base to make one suspect that there might be pressure on some part of the bronchi of that lung. At that time the blood was—reds 6,080,000, hæmoglobin 90, colour index .75, whites 5,700, polymorphs 70, lymphocytes 25, and eosinophils 5. He was then taken into Chalmers' Hospital, and an X-ray photograph was at once taken. It was found that in addition to the superficial glands, there was a considerable mass to be seen in the mediastinum. We resolved to try the effect of X-ray treatment, and of radium, and

Dr Dawson Turner, at the Infirmary, kindly undertook this for us. Unfortunately, his admission to the hospital was immediately followed by a febrile wave, which lasted nine days. As soon as this had stopped, the treatment was commenced, and continued every second day for ten days, during which the temperature remained normal. During this febrile period, when the glands were distinctly swollen and tender, a hypodermic needle was pushed into the axillary mass, and part of the fluid withdrawn was used to make a culture, which, however, showed nothing but ordinary skin organisms. During the febrile period the leucocytes steadily increased, reaching 13,000 at the height, and falling to 6,000 when the fever was over. There was a definite polymorph increase, the proportions being, polymorphs, 80 per cent., lymphocytes, 20 per cent. This leucocyte rise and polymorph increase occurred with each of the attacks of fever afterwards. By about the beginning of January we saw that it would be hopeless to continue the X-ray treatment if the attacks of temperature were to recur, as he was much prostrated by them. We were experimenting with salvarsan in pernicious anæmia at this time, and it occurred to us that it might be worth trying in this case also. On January 4th, he received .4 grams in the right buttock. There was no febrile reaction, not much pain, but the glands in the neck and axilla diminished slightly in size. At the usual time, however, he had a fresh rise of temperature, which attempted to come down to normal at the end of ten days, but passed on into another series of waves, lasting until the 7th of February, the temperature being much more irregular than before, and swinging between 99° and 103°. During that period one of the axillary glands was excised and was found to show the typical appearances of soft lymphadenoma. Various remedies were used to reduce the temperature. We tried anti-streptococcous and anti-staphylococcous serum, without effect. Cryogenin in 5 grain doses was the only thing which had much effect in reducing the temperature, it certainly relieved his symptoms. On February 7th we gave .6 grams of salvarsan intravenously. The patient had a rigor an hour later, and on the same evening a little sickness and slight jaundice, but he was not so much disturbed as, for example, a true syphilitic case would have been. There was no albuminuria. The effect of this injection was most striking. The temperature fell to normal practically at once, and remained so, the glands shrivelled up into hard, small lumps, his appetite returned, and during the time that he remained in hospital there-

after, until March 11th, that is to say, he put on 10 lbs. in weight, and recovered his look of health. By that time he seemed so well that he was sent home. He had only been at home four days when his temperature again rose, the glands became tender, and by his own wish he came back to hospital on March 28th, in order that he might have the injection repeated. He was given .6 grams of salvarsan on March 30th ; there was a slight rigor, slight sickness and diarrhœa, and a little injection of the conjunctivæ. As before, the temperature fell, and the glands which had been swollen, quickly became smaller. The effect, however, on this occasion did not last so long. On April 8th the temperature rose, and the glands became large and tender. We were afraid to give the salvarsan intravenously again, after so short an interval, and gave him .6 grams in the right buttock, without, however, there being the slightest effect on his temperature or on the glands, while there was a good deal of pain and swelling at the seat of injection. This febrile attack went on until the 8th of May, with steady increase in the size of the glands, emaciation, and the usual febrile symptoms. We tried giving quinine intravenously, and various other remedies, but without any effect on the temperature. On May 8th he received .4 grams of salvarsan intravenously. On the following day the temperature fell below normal, the leucocytes, which had been raised, fell to 5,000, the swelling of the glands diminished. Temperature remained normal for four days, then rose, and after three days of fever, on the 16th May, he received .4 grams again. The temperature again fell, but remained down for two days only, and he gradually developed jaundice and broncho-pneumonia, and died on the 29th. The post-mortem examination was performed on the 30th, and the masses of glands which had been obvious in the left neck and axilla, were found to be the ends of a chain running through the mediastinum, and connected with a chain of smaller glands running down the aorta to its bifurcation. There were a few small recent glands in the right axilla, the liver showed a few flat nodules on its surface, nothing in its substance, the spleen was not much enlarged but contained numerous nodules from the size of a barley-corn to a pea scattered through it. There was nothing elsewhere. The only trace of tubercle in the body was one or two calcareous glands in the mesentery which had not been affected by the lymphadenoma at all. There was also a healed ulcer in the bowel, which might possibly have been tubercular. On microscopic examination of the glands, they were found to show

the typical appearances of lymphadenoma, the smaller and more recent ones being "soft," the older "hard." No giant cells were found anywhere; there was no excess of eosinophils in the glands or elsewhere.

The special point of interest in the case is the extraordinary effect which the salvarsan had on the course of the temperature, on the size of the glands, and on the patient's general condition. The first remission produced in this way lasted over a month, and at the end of that time the patient looked perfectly well, his glands were hard, small, and gave him no inconvenience, and one almost hoped he was on the direct road to recovery. It looks as though we had just missed a success, and in dealing with future cases of lymphadenoma it might possibly be worth while to treat them with intravenous injections of small doses of salvarsan at regular, say monthly, intervals, in the hope of checking the progress of the disease. It was quite evident to us that the intramuscular injection had no effect in the way of producing improvement, and this has, indeed, been our experience in a large number of other cases of different diseases.

The *President* said he supposed it was because the case did not admit of any discussion that none of the members had spoken. The case raised a very interesting point. Hitherto, as every one knew, arsenic was the only remedy of use in lymphadenoma, and it would be exceedingly interesting to see if the remarkable improvement which Dr Gulland had reported in this case is verified in future cases. The temporary success of the treatment certainly warranted one in trying it in future cases of the disease.

V. Discussion

THE PART PLAYED BY THE UPPER AIR PASSAGES AS AVENUES OF GENERAL INFECTION

Introduced by A. Logan Turner, M.D., F.R.C.S.E.

The subject selected for discussion this evening covers so wide a field that it would be impossible in the time at our disposal to deal with it in a thoroughly comprehensive manner. Nor would it be wise to do so, if discussion is to be stimulated. The subject must necessarily be of intense interest not only to every general practitioner of medicine, but to the specialist in every branch, to the Public

Health official, and to the pathologist and bacteriologist. Our purpose therefore is to open the discussion along certain lines and to ask the members present to develop it according to their own personal experience. By the upper air passages we mean the nasal cavities with their accessory sinuses, the naso-pharynx with its diverticula, the middle ear clefts and the pharynx. The gums and the teeth cannot be regarded as coming into the definition of upper air passages. The reasons which render this gateway so prone to the introduction of infective material into the organism are sufficiently obvious. As respiratory passages, and over a limited area as part of the alimentary canal, they are open to both air and food infection. Further, both in health and in the catarrhal conditions of the mucous membranes of individuals apparently in the enjoyment of excellent health, micro-organisms of various kinds are constantly present: amongst such may be mentioned the various pyogenic cocci, the pneumococcus, the micro-coccus catarrhalis, organisms of the diphtheria group, the influenza bacillus, the meningo-coccus and the bacillus tuberculosis. Those organisms vary in number, in vigour and in pathogenicity, while from time to time the tissues of the host from various causes may undergo a diminution in their resisting power, or fresh organisms may gain entrance, and bacteria, previously quiescent or avirulent, may become active and give rise to acute infections. Again we find in the mucous membrane of the upper air passages a well developed lymphatic network, and free communication by efferent vessels with the cervical lymphatic system and through it with the blood stream. The experiments of Ribbert and Roth, Schimmelbusch and Lexer have demonstrated the readiness with which a general blood infection may follow the smearing of the pharyngeal mucous membrane with the streptococcus. Lexer's experiments are particularly interesting as showing the rapidity with which a general blood infection follows this method of inoculation. In animals dying twenty hours after infection of the pharynx with streptococci, a pure culture of the same was found in the blood in $3\frac{1}{2}$ hours. The local appearances of the throat confirmed the view that the infection had spread from the seat of inoculation: redness and swelling of the mucosa, with difficulty in swallowing and swelling of the cervical lymphatic glands being evidence of local changes.

The pathways of infection from this area are numerous:—

(*a*) The lymphatic ring with the naso-pharyngeal, faucial, and lingual tonsils and the lymph nodes of the pharynx, all communicat-

ing with the cervical lymphatic system, and thence with the blood circulatory system.

(*b*) The venous channels, connecting the upper air passages with the central nervous system.

(*c*) The lower air passages in direct continuity, and hence inhalation infections.

(*d*) The œsophagus in direct continuity, and hence ingestion infection.

The subject might be dealt with from the side of the organism, but I have selected for its consideration an anatomical basis, as indicated in the following headings.

A. Infection of the Cerebro-spinal System

(*a*) *The Pyogenic Organism.*—The occurrence of meningitis, sinus thrombosis and brain abscess, as secondary to suppuration of the nasal and accessory sinus cavities and of the middle ear cleft, is too well known to require special reference. Nevertheless, it is necessary to draw attention to it, because our experience is that there are a number of cases, especially cases of lateral sinus thrombosis, in which the diagnosis of the primary source of the infection is overlooked. It is possible that no reference is made by the patient to aural symptoms, while the medical attendant may fail to make the necessary inquiries. While this is true of latent middle ear suppuration, it is even more so in connection with suppurative disease of the posterior ethmoidal and sphenoidal air sinuses, because the nasal discharge may be too insignificant to call for any remark. In the great majority of cases of accessory sinus and ear disease the infection spreads by continuity of the inflammation through the contiguous bony walls, in many instances the bone becoming carious or completely absorbed. In other cases infection takes place through the venous channels which effect a communication between the upper air passages and the interior of the skull. The question of infection by way of lymphatic channels requires further elucidation. A careful inquiry into this possible avenue of infection has led me to the conclusion that proof of the same is still wanting. Cuneo and André have been able to inject in young human subjects a lymphatic communication between the meningeal spaces and a network upon the olfactory part of the nasal mucous membrane, the lymphatics traversing the cribriform plate by small canals. Flatau

was able to inject the lymphatic vessels of the naso-pharynx from the subarachnoid space. We have been unable to find in any of the pathological reports made upon persons dying from intracranial complications secondary to nasal and accessory sinus suppuration histological evidence of the spread of the infection by way of lymphatic channels.

(*b*) and (*c*) *Pneumococcal and Influenzal Meningitis.*—Conclusive evidence has been brought forward that pneumococcal and influenzal meningitis may arise as a direct infection from the upper air passages. We have only to refer to the investigations of Weichselbaum, Pfühl, Grober, E. Fränkel, Ghon and others who have demonstrated the pneumococcus and the Pfeiffer bacillus in the accessory sinuses and middle ear cleft, and also in the meningeal exudate in cases in which the other organs of the body were healthy. In connection with (*d*) *Epidemic Cerebro-spinal Menengitis* many observations might be quoted as to the occurrence of the meningococcus in the naso-pharynx, not only in those who have been the victims of the disease, but also in those who have been in contact with patients, and who must be regarded as carriers. The investigations of Westenhöffer and Lingelsheim demonstrate that the naso-pharynx is a more frequent portal of infection than are the nasal cavities and accessory sinuses. E. Meyer examined a number of children affected with the disease in Upper Silesia, and during the early days of the affection well marked inflammatory changes were observed in the naso-pharynx. These observations have been confirmed by Busse. If these facts are to be accepted, by what pathway does the organism reach the meninges? The part played by the lymphatic vessels is still open to discussion. Possibly in some cases infection may pass through the sphenoidal sinus and thus reach the meninges, but Hirsch has raised the question as to whether the middle ear cleft may not sometimes be the route of infection, and thence through the labyrinth and internal auditory meatus. This suggests that the labyrinthine deafness of cerebro-spinal meningitis may not always be secondary to the meningitis, as has so long been held, but may possibly precede the meningitis.

With regard to *acute poliomyelitis*, Flexner considers that all the theoretical conditions required to establish the naso-meningeal route of infection have been supplied by experiment. Although the etiological factor in poliomyelitis has not been demonstrated microscopically, Osgood and Lercas have demonstrated the possibility of transmitting from monkey to monkey a typical poliomy-

elitis from the filtrate of the naso-pharyngeal mucosa of two monkeys dying without any other obvious infection at an interval after the acute stage of the disease. It has been shown that naso-pharyngeal catarrh is very prevalent in communities in which poliomyelitis exists. Wickham has called attention to the lymphatics as the route of infection. In all probability further light will be thrown upon the mode of spread of this disease in the immediate future.

(*e*) *Tuberculous Meningitis.*—Considerable discussion has from time to time taken place as to the part played by the nasal and post-nasal cavities and their lymphatic tissues as portals of infection in tuberculous meningitis. Weigert believed that this was the usual gateway of infection. The existence of tubercle bacilli in the healthy nose, their presence in adenoid vegetations and in middle ear disease, the occurrence of primary nasal tuberculosis as a more common affection in children than was at one time realised, and the close proximity of the nasal and cerebral cavities would all point to the upper air passages as a frequent portal of infection.

B. Infection of the Lungs: Pulmonary Tuberculosis

Probably in a discussion of this kind interest will centre mainly round the question of the pathway of infection to the lungs by the tubercle bacillus. The problem of ærogenic or inhalation invasion as against lymphatic infection: in what proportion of cases are the lungs infected by the inhalation of the tuberculous virus, or through the cervical lymphatic system from inoculation of the mucous membrane of the upper air passages? This leads us to a consideration of the anatomical arrangement of the lymph drainage of this area. Briefly, the whole of the lymphatic drainage of the nasal, naso-pharyngeal and pharyngeal mucous membranes passes through the deep cervical glands into the large veins at the root of the neck and thence to the right side of the heart and pulmonary artery. The tracheo-bronchial glands receive their afferent vessels from the mucous membrane of the trachea, bronchial tubes and pulmonary alveoli. The efferent vessels from these glands terminate in a broncho-mediastinal trunk which opens into the large veins at the root of the neck. Anatomically, no intercommunicating lymphatic vessels have been demonstrated either between the inferior deep cervical glands and the tracheo-bronchial glands, or between the latter and the deep cervical glands. Hence, anatomically, a descending tuberculous invasion from the cervical to the peribronchial glands, or an ascending infection from the latter to the

cervical glands, is not possible. Clinically, however, there is evidence that the deep cervical (supra-clavicular glands) can be infected from the mediastinal glands. This is seen in the case of malignant tumours of the mediastinum, and one could quote cases in support of it.

Experimentally, it has been shown by many investigators that inoculation of the mucous membrane of the upper air passages with the tuberculous virus is followed by infection of the cervical glands and the development of pulmonary tuberculosis. Clinically, too, we have evidence of the entrance of the tubercle bacillus into the lymphatic ring, and the results obtained by numerous investigators are recorded upon the tables on the wall; these figures deal only with cases of primary tuberculosis of the tonsils, and show in the case of the naso-pharyngeal tonsil an average of 6% harbouring tubercle with a variation from 2 to 20%, and in the case of the faucial tonsil an average of 8% with a variation from $\frac{1}{2}$ to 15%. We have also clinical evidence of the infection of the cervical glands in many of these cases. Having demonstrated the entry of the bacillus through the mucous membrane into the glands, can we always determine the cervical glands as the pathway of infection to the lungs? From the upper air passages three pathways of infection are possible—inhalation, ingestion and the cervical lymphatic system. Tubercle may enter by all three avenues in the same individual, or by two, or by one of them. The inhalation experiments of Louis Cobbett are of great interest in this connection. By spraying emulsion of tubercle bacilli in front of guinea pigs he found that when the animals were killed, after a few minutes or hours, the lungs were affected, but the cervical glands showed no evidence of tubercle. On the other hand, in seventeen animals similarly dealt with, which were allowed to live from five to thirty-six days, sixteeen showed pulmonary and bronchial gland tubercle, while in thirteen the deep cervical glands were also affected, their infection taking place concurrently with, but more slowly than, the lung infection. The lungs were not infected through the glandular chain, but by inhalation. Another difficulty met with in attempting to follow the path of infection is this, that the tubercle bacilli may leave no sign to indicate their point of entry, and they may be present in the lymphatic glands without producing any histological evidence of their existence there. Bartel and Spieler amongst others have clearly demonstrated this point. Dr Mitchell, who has kindly examined some of my cases, has obtained four positive inoculation results out

of six tonsils experimented with; in two of the positive inoculation results there was no microscopic evidence of tubercle. Again, it must be remembered that once the lungs have become the seat of tubercle, the tonsils and cervical glands may be secondarily infected by the tubercle laden sputum passing over the pharyngeal mucosa. In the investigations, the figures show that tubercle may be found in the tonsils of phthisical patients in 71% of the cases, the percentage varying from 33 to 100%. These figures show a striking difference to the percentage met with in primary tonsillar tubercle, and afford ample proof of how frequently the tonsils may be secondarily infected. Lastly, tubercle bacilli may be swallowed in naso-pharyngeal and pharyngeal secretions, and the disease thus reach the mesenteric and other groups of glands. The difficulties in demonstrating the exact pathway of infection are therefore obvious.

A point which one would like to obtain some assistance upon during the discussion is, in what proportion of our cases of tuberculous cervical glands do we find pulmonary tuberculosis developing? Judd, working in the Mayo's clinic, has recently analysed 754 cases of partial and complete excision of cervical glands: ten of these cases had pulmonary tuberculosis at the time of the operation, nineteen have since died of the same disease, and nine from tuberculosis elsewhere in the body. What is the experience of other surgeons?

In our experience, which is entirely clinical, it is rare to find pulmonary tuberculosis developing in connection with primary tuberculous disease of the nose and pharynx. I have had an opportunity of examining fifty-six cases of primary nasal and pharyngeal tuberculosis of the variety known as lupus, admittedly a slow form of tubercle containing few bacilli. The disease persists locally for years; the upper deep cervical glands become enlarged. I have only knowledge of two cases in which phthisis pulmonalis developed. In cases of laryngeal tuberculosis secondary to pulmonary phthisis one does not detect enlarged cervical glands. While evidence of tubercle is found from time to time in our cases of adenoids and tonsils, and the cervical glands are enlarged, I have at the present moment notes of only one child in which at the same time there is tubercle of the lung apices. Inoculation of the tonsils, by Dr Mitchell, into guinea pigs revealed a mild tuberculous infection, but no tubercle was detected microscopically in the tonsils. One has no means of determining whether the pulmonary condition

is of lymphatic origin or has resulted from inhalations, or the cervical condition is secondary to the pulmonary disease ; or have the two conditions gone on *pari passu* ?

Granting, however, the cervical lymph glands as an avenue of infection, how does the tubercle bacillus pass from them to the lungs. Three propositions have been put forward ; first, the one based upon the anatomical arrangement of the cervical lymphatics already described, namely, by way of the large veins at the root of the neck, the right side of the heart, and thence by the pulmonary artery to the lungs and tracheo-bronchial glands. Secondly, the theory of Klebs, Grober, Philip and others that the lowest deep cervical glands infect the apex of the lung by direct extension through the tissues, or, as suggested by James Ritchie, the bacilli may be drawn into the apex of the lung from the infected glands by a process of suction from the respiratory movement of the chest. Thirdly, the view based on the assumption that an anatomical connection exists between the deep cervical and tracheo-bronchial glands, so that infection passes direct from the former to the latter and thence by reversal of the lymph current to the lungs. Time will not permit me to review these various points, but we may state here that the view that the apex of the pleura and lung is directly infected from the cervical glands has been strongly opposed. What evidence is there at autopsies upon patients dying of pulmonary tuberculosis of adhesions between the glands and the apex of the lung ? How often are tuberculous glands found in the neck in these cases after death ? Walsham found in a considerable number of cases, in twenty-six out of twenty-seven, associated cervical gland and pulmonary tubercle at autopsy. Were the glands secondarily infected from the sputum ?

In connection with the cervical lymphatic gland pathway in the pathogenesis of pulmonary tubercle, I would like to quote the conclusions drawn by Beitzke from a number of post-mortem examinations :—As pulmonary and bronchial gland tuberculosis is usually of longer standing and has made more progress than that in the cervical glands, the former have been infected by inhalation of the virus, while the latter have become infected either secondarily by inoculation with tuberculous sputum through the tonsils, or independently of the lung disease but co-existent with it and from the same source. Further, that in the absence of any direct lymphatic connection between the cervical and bronchial glands, tuberculous infection of the lungs by way of the cervical glands can

only take place through the large veins and the right side of the heart. Albrecht, from a study of 1060 tuberculous children, has formulated the view that the primary site of infection is most often in the lung from inhalation. If there is also tuberculosis of the cervical lymphatic glands, it is secondary to the pulmonary affection, either from secondary infection of the pharynx through the sputum or by an ascending gland infection from the peri-bronchial glands upwards. There is no doubt that in connection with the whole of this subject of tubercular infection of the lungs, further investigation and observation are necessary. Perhaps the discussion this evening may throw further light on the subject.

C. Infection of Joints and Bones

(*a*) *Acute Rheumatism.*—Many facts point to the infective nature of acute rheumatism, and during recent years the possibility of the throat as the portal of infection has been much discussed. Many writers have dealt with the subject and described an acute inflammation of the tonsils as preceding the joint affection. Poynton was able to isolate from the tonsil the same organism which he had already succeeded in obtaining from an endocarditis, pericarditis and arthritis. Cases of endocarditis have been reported as occurring during the course of acute tonsillitis and the possible rheumatic character of these cases of endocarditis has been discussed. The valvular inflammation may precede an affection of the joints, or the arthritis may occur independently. The reports of Gürich and Schichhold upon the treatment of the tonsils in rheumatic affections are interesting in connection with this portal of infection. Gürich cured 98 out of 125 cases of articular rheumatism by removal of the tonsils, while Schichhold had similar results in 70 patients. In a great many of these cases the tonsils were not inflamed, but on superficial examination presented a normal appearance. More careful examination of their crypts revealed the presence of inspissated secretion. The authors regarded the rheumatic pains as induced by the toxins absorbed from the organisms contained in the crypts. I can bring no personal experience of any value upon this question, but the observations of the physicians present would prove of interest to us.

(*b*) *Suppurative Affections of Joints and Bones.*—That pyogenic organisms may reach the joints and bones, the infection entering through the lymphoid tissues of the upper air passages, must be definitely accepted as proved. We have already referred to Lexer's

experimental work and the rapidity with which the blood may become infected after inoculation of the pharynx. In all probability those rapid cases of bone and joint infection, in which no intestinal or pulmonary affection exists as a primary source, can be explained in this way. Buschke describes a case of simple fracture of the humerus in which a suppurative periostitis supervened three weeks after the accident. At that date the patient developed an acute tonsillitis; from the tonsil crypts a pure culture of streptococcus pyogenes was obtained; two days after the onset of the angina a pure culture of the same organism was obtained in his blood. On the following day the patient had a rigor, with pain at the seat of the fracture. After a second rise of temperature fluctuation was detected, and an abscess was opened and a pure culture of the streptococcus was obtained from the pus.

The same author describes a case of acute ostomyelitis in a boy aet. 14, who, three days after an acute tonsillitis, suffered from pain in, and swelling of, the tibia and foot. A large abscess was evacuated; at the time of the operation the throat was still inflamed: the staphylococcus aureus was cultivated both from the tonsils and from the abscess. In this case the blood was not examined. An illustrative example of an acute suppurative arthritis quite recently came under my own observation. The patient was admitted with chronic middle ear suppuration and symptoms of sigmoid sinus thrombosis. Operation was performed, the breaking down clot removed and the internal jugular vein which also contained the clot was tied. A pure culture of the staphylococcus aureus was obtained from the clot in the sinus and from the blood in the arm. The patient progressed satisfactorily for a fortnight, when he developed an acute tonsillitis with secondary laryngitis. A severe general infection followed and an abscess formed in the left knee joint. A pure culture of streptococcus pyogenes was obtained from the pus. Unfortunately the blood was not examined at the same time. At the post-mortem the intracranial sinuses were found normal and there were no secondary lung abscesses. Death had resulted from the more recent acute streptococcal infection. These cases illustrate sufficiently this aspect of our subject.

D. Infection of the Intestinal Canal

Appendicitis.—In the various discussions which have arisen in connection with the origin of appendicitis, the possibility of an

etiological relationship between tonsillitis and appendicitis has been considered, as giving a possible explanation of some, at any rate, of these cases. Kelynack appears to have been the first to draw attention to this association and noted the occurrence of an acute gangrenous appendicitis during the early stages of an acute tonsillitis, which closely resembled diphtheria. Since then numerous writers have drawn attention to this aspect of the subject. In two cases published by Kretz the streptococcus pyogenes was obtained both from the tonsil and from the peritoneal exudate. The question of the pathway of infection from the throat to the appendix has also been discussed, some favouring a blood infection, others infection through the gastro-intestinal canal. In a recent paper by Bachhammer, from the Pathological Institute in Munich, dealing with the relation of the tonsils to general infections, it is interesting to note that out of thirty-seven cases of tonsillitis with subsequent infection of other organs, eight cases of acute appendicitis are included in his list. Unfortunately no bacteriological examination appears to have been made. The writer is careful to observe that these cases form only a small proportion of the many cases of appendicitis which occurred during the three years which his observations covered. George Sutherland has discussed this subject in a way which is specially interesting to us this evening, namely, the association of appendicitis with rheumatism and its relation to the faucial tonsil. The appendix with its lymphoid tissue, described by Bland Sutton as the " abdominal tonsil," has been regarded in its healthy state as performing a function similar to that of the faucial tonsil, and under certain conditions it is reasonable to suppose that its function may be impaired or destroyed and that, like the faucial tonsil, it may become liable to inflammatory attacks. Sutherland raises the question whether such impairment may not take place in connection with the presence of the rheumatic virus in the circulatory system. If the organism of acute rheumatism gains entrance through the faucial tonsil, and if the lymphoid tissue of the appendix may be impaired in function in association with the rheumatic virus in the blood, then we can see how the faucial tonsils stand in relationship to appendicitis.

E. General Septic Infections

Under this heading I wish to refer to some cases of very considerable practical importance, in which the possibility of the existence of the primary focus of infection in the upper air passages is apt

to be overlooked. These cases have come under my own observation. The nasal or pharyngeal condition may be so insignificant that neither the attention of the patient nor that of his physician may be called to it. The similarity of the symptoms in one case to malaria, in another to typhoid, and in another to pulmonary phthisis adds additional interest to the study of such cases. In one case the patient suffered for four weeks from an evening rise of temperature, reaching 101° and 102° F. The morning rise was less marked. She complained of night sweats. Frequent examination of the chest failed to detect any signs of tubercle. Examination of the nose revealed a collection of muco-purulent secretion in the naso-pharynx. The accessory sinuses were healthy. Daily syringing of the nose was commenced, and rapid improvement followed, the temperature gradually falling to normal and remaining so. In another instance, a patient complained of nervous irritability, cold extremities, rapid pulse and flushings. Though he never complained of his nose, examination revealed a collection of foul-smelling pus in the antrum of Highmore—after opening and draining of the sinus all his symptoms disappeared. The chart on the wall illustrates a case of acute general streptococcal infection, a pure culture being obtained from the blood on three different occasions; the patient complained a great deal of his throat, and the naso-pharyngeal and pharyngeal-mucous membranes were constantly bathed with secretion. In cases of ozæna symptoms of general septic infection may be met with. In one case the occurrence of rigors, with temperature rising to 103° and 104° F., was explained from this cause. The patient kept a thermometer, and recorded his own observations. The authenticity of his statements were proved, as on two occasions while he remained in hospital under observation similar rigors were noted. An interesting point was noted in this case: if the irrigation of the nose was not carried out in the morning but delayed until later in the day, a rigor was a not infrequent sequel. In connection with the existence of adenoids and enlarged tonsils, I have notes of three children who suffered at intervals from attacks of bilious vomiting. Since the removal of the adenoids and tonsils, these attacks have entirely ceased. Last year I removed the adenoids and tonsils from a child who for six weeks had a daily rise of temperature. Tubercle was suspected, but nothing positive could be detected. Since the operation the temperature has remained normal. No one can gainsay the practical interest of such cases, which certainly point to the value of examining the upper air

passages in septic conditions of doubtful origin. While the above notes indicate the importance of the upper air passages as gateways of general infection, they do not exhaust the subject: they may, however, serve as a suitable text for introducing a discussion.

The *President* was sure the members would agree that they were very much indebted to Dr Logan Turner for the extremely interesting and lucid way in which he had brought this subject before the Society. It was a subject of very great importance and wide interest; it interested both the ordinary physician and surgeon and the scientific pathologists; no doubt there would be a good discussion and very valuable information obtained.

One point Dr Logan Turner had not mentioned, namely, the protective influence of tubercle in the glands of the neck against the production of tubercle elsewhere. As regards the question of rheumatism the general practitioners would be able to give their practical experience; and he hoped the pathologists would also give their views as to the channels of infection.

Dr Philip agreed with the President as to the admirable nature of Dr Logan Turner's remarks; he had introduced them with a lucidity and anatomical ability which recalled his ancestry in a very pleasing fashion. This was perhaps the most interesting and significant discussion which had taken place before this Society. Curiously enough, so far as he (Dr Philip) had observed, Dr Logan Turner had made no reference to local growths—diphtheria, scarlatina, measles, etc. Although the etiology and pathology of these diseases had not been established, it was significant that they had for their primary manifestation local disturbance in that region. It was the consideration of facts like these and of the frequency with which patients whose histories are carefully investigated reveal statements regarding throat lesions that led him to look very carefully into the tonsils and tonsillar region as the paths of entrance for tuberculosis.

In order to make an exhaustive study of this subject it was necessary to commence with the child, more particularly in connection with tuberculosis; if one waits to investigate the subject in adults a number of fallacies are met with which are avoided in the child. With regard to the glands themselves, changes occur during the development of adolescence which obscure the issue very con-

siderably. His view, speaking broadly regarding the introduction of tuberculosis, is as follows :—

Tuberculosis may enter the system practically at any point either of the respiratory or alimentary tract ; but perhaps the most vulnerable point, as Dr Logan Turner pointed out, is just where the two tracts cross each other. There you have the possibility of contamination from two passages and you have a certain amount of stasis. The child's mucous surface is much more absorbent than that of the adult. Further, the child, in virtue of its environment—close confined rooms, etc.—is in a more or less devitalised condition ; and because of its more or less dirty habits is exposed to contamination from other sources. The entrance of the tubercle bacillus is thus made every easy. It must be a matter of common observation that enlargement of the extra-tonsillar gland is frequently found ; that has led to further observations. He believed that in the majority of cases in which there is enlargement of the extra-tonsillar gland it is due to tuberculous infection ; whether this is so in all cases he was not prepared to say. The progress of these cases warrants this conclusion. The condition goes on until the glands at a lower level begin to enlarge, until finally there is a fan-like arrangement of the enlarged glands, and the same process is going on in the deeper glands as in the superficial. One has been able to follow the infection through the supraclavicular triangle and subsequently to the lung apex. The clinical observations are highly suggestive ; it must be remembered, however, that these clinical observations have been substantiated in large part by experimental observations. Woodhead and many other observers have shown that manifestations of tuberculosis can be produced by feeding pigs with food containing tubercle bacilli. The first indication is enlargement of the glands in the region of the angle of the jaw, then follow successive enlargements of the glands throughout the cervical region, both superficial and deep ; quite early pleurisies are established with apparently minor adhesions between the masses in the pleura, and therefore tuberculosis is passed to the lung. If that is so in the gland of pigs, why not in the human subject ? Further, as Dr Logan Turner has pointed out, the experimental evidence goes clearly to show that in a large percentage of cases the cervical glands do contain the tubercle bacilli in large amount.

With regard to the point raised as to the relationship between a tuberculous infection of the superficial glands and the frequency of

tuberculosis, he referred to a fallacy which has caused a great deal of trouble, namely, that the tuberculous cervical gland which is most important is the deformed suppurating gland; that he (Dr Philip) believed to be the least serious of all; it is a manifestation of tubercle, but it is associated with recovery. It is the small infiltrating gland which seems capable of passing on the tubercle bacillus elsewhere. It is a gradual process of inoculation by one of the extra-tonsillar glands through this fine mesh-work of glands which goes on until the proximity of the apex of the lung is reached. That he believed to be the common pathway of tubercle. If that be so it emphasises the great need of systematic preventive measures with regard to the throat. Having regard to the facts elicited regarding the teeth of the nation, and knowing that the same conditions which influence the teeth are at work in relation to the throat, one of the most important aspects of preventive medicine is the regular toileting of the throat. One has been impressed while watching the children of tuberculous patients with the fact that in proportion to the manner in which attention to the throat is carried out, tuberculosis is limited.

Mr Scott Carmichael was greatly interested in the paper, but Dr Logan Turner had raised so many points it was difficult to know where to begin. Infective glands in the upper part of the neck in children were found in three situations, namely—(1) in a definite area between the posterior belly of dyastric, internal jugular and common facial veins; (2) round the spinal accessory; and (3) along the posterior border of the internal jugular vein. In children under one year old, the glands most frequently affected are those which drain the middle ear and mastoid antrum situated round the spinal accessory; so constantly is this the case that when these are affected in a baby under nine months or one year, one looks for the primary disease in the Eustachian tube, the middle ear or the mastoid process. When a child is brought with this condition, without any discharge from the middle ear or any swelling over the mastoid, one can be almost certain that there is extensive disease of the bone of the mastoid process. In these cases he thought the mastoid process should be cut down upon because diffuse tubercle was frequently to be found. The infection usually has taken place by the Eustachian tube. Another point is that these children are those who have been bottle-fed; he had never seen a case of this kind in which the child had been fed

on the breast—one almost need hardly ask the mother how the child has been fed.

In older children the tonsillar gland is affected more than any other ; this receives its lymphatic supply from the tonsil. Out of 50 cases in older children, 7 showed evidence of tuberculosis on microscopical examination. The small tonsils were found to be affected, not the large ; that is an interesting fact. At the same time he had also examined a number of hypertrophied tonsils, but found no tubercle. The tubercle generally began at the bottom of the crypts and extended throughout the whole, causing fibrosis and shrinkage of the tonsil. It was impossible on clinical examination of the throat to say that there was any tubercle. The tonsil was, he thought, the primary focus and justified the removal of the tonsil in every case whether it is enlarged or not. Another point is that these glands are infected the one from another ; the infection begins in the tonsillar set and spreads to the other two sets. It is extremely important to remove all three sets of glands even although there is no clinical evidence of tubercle ; he did not think the surgeon was doing his duty unless he did so. He mentioned a case of pneumococcal infection of the mastoid, where a child, under one year, subsequently developed pneumococcal peritonitis, pneumococcal arthritis, and pneumococcal septicæmia in the course of a day or two.

With Dr Philip's statement about direct infection to the lung from the glands of the neck, he could not agree. He had dissected a large number of glands and had never seen any evidence at the operation that there was any infection of the apex. In the 50 cases already referred to in only one was there any evidence of lung disease. Where one finds extensive glandular disease, one does not find lung infection.

Dr Fowler said about eighteen months ago Dr Philip came over to the Hospital and demonstrated these glands, which are extremely common in children and very interesting. Previously he had regarded tuberculosis of the lung in children as a comparatively common phenomenon, but it was not a thing one had paid particular attention to. Since Dr Philip's visit, however, he had looked for these glands and found them in a very large proportion. But when it comes to post-mortem examination one has difficulty in finding that these glands are tuberculous. By the naked eye it is not possible to say that they are not tuberculous. Certainly

the small glands that Dr Philip speaks of were not found by him to be of a tuberculous nature.

Dr Hunter wished to add his testimony to the extreme value of Dr Logan Turner's paper. Not only had it been presented to the Society from a scientific point of view, but also from the anatomical and clinical points of view. The point he wished to refer to was with regard to Mr Scott Carmichael's statement referring to the infection of the apex from cervical glands. He (Dr Hunter) had always been much alarmed, and still was, on finding enlarged cervical glands, and he had looked forward with apprehension, and was not yet free from apprehension, to the possibility of the lungs becoming involved ; but after hearing the extremely exact anatomical definition given of the channels of infection from gland to lung, he began to think that the possibility of that infection was not so great as he had believed. His reason for rising to speak was to ask some of the anatomists present to give the Society the benefit of their special knowledge on the source of infection of the lower cervical glands and the avenues of infection to the apex of the lung.

Mr Cathcart did not think it mattered whether one could trace any anatomical connection between the glands and the apex of the lung. Cases of tubercle in the joints were constantly met with where undoubtedly the infection must have been carried to the lung. In cases of tuberculous disease of the joints almost invariably there is some focus in the glands, either the cervical or mesenteric glands. He had always felt when dealing with tuberculosis of a joint that it must have come through the alimentary or respiratory tract. If that is true of the joint it must be true of the lung. Practically, he did not think that the anatomical relationship between the gland and the lung is of very much account. His general observation seemed to show that tuberculosis of the gland is seldom associated with lung involvment. He had frequently seen masses of gland right down on to the clavicle where there was no infection of the lung.

Then Mr Scott Carmichael raised the question of excision of the tonsil in cases where there is tuberculosis of the cervical glands. He (Mr Cathcart) had always felt that one did not do one's duty by merely excising the gland ; he always gave directions that the patient's throat should be thoroughly well painted, and if there is anything found manifestly wrong with the tonsil to have it treated.

He asked Dr Logan Turner if he thought that the tonsil might recover when it had been contaminated by the gland? His own feeling was that the tonsil did not become infective and therefore it was unnecessary to deal with it. As to whether the surgeon ought to take away all the glands in cases where he has detected a limited tuberculosis of the cervical glands, his own opinion was that lymphatic glands are naturally protective; and also there is a great power of recuperation in tuberculous glands; consequently all that was necessary was to clear away all the definitely affected tuberculous glands. Some surgeons strongly urge the removal of the whole of the glands, but he (Mr Cathcart) always held that it was a safer thing to leave them, even although there was a recrudescence with the greater part of the glandular tissue, so long as it was not necessary to remove them.

Dr Shennan had had some experience of these cases in the Sick Children's Hospital and also since. Dr Turner had not referred specially to the retro-pharyngeal gland, which drains the posterior part of the nasal cavities. He had been looking for these retro-pharyngeal glands recently; he had not now the same opportunity of examining children, but in the adult it is very difficult to find these glands affected. The question comes to be whether these glands escape. In very young children there is the possibility that the petrosal squamous suture is not closed and affords another channel for the organisms passing up to the Eustachian tube.

With regard to the tonsils his experience pretty well coincided with that of Dr Logan Turner, and also with regard to spreading down to the bronchial glands. From his post-mortem experience in the Sick Children's Hospital he had found that while the glands in children are very extensively affected, and the mediastinal glands affected in perhaps the majority of cases, the glands at the root of the right lung are very much more affected than those of the left lung. He had never been able to explain this fact. About the same time as he had made this observation, Dr Still observed the same thing. Sometimes the root of the left lung is also affected, but it is usually the right lung in a much greater degree.

With regard to whether the absence of evidence of infection of the tonsil indicates that the infection is not through the tonsil, one cannot go by that at all; one knows that in other parts of the body sets of glands can be skipped.

With regard to infection of the lung. In these cases it is quite

the exception to get tuberculosis of the lung. One considered the glandular infection was primary, except in cases where there was tubercle before. As regards the tonsil in pyæmia, in so-called cryptogenic pyæmia, one has got into the habit of examining the tonsil because the infection may be from the tonsil. In typhoid, too, the infection may be derived from the tonsil.

Dr Chalmers Watson confirmed what Dr Logan Turner had said as to the occurrence of gastric and biliary disturbances arising primarily from infection of naso-pharynx. He also referred to nephritis, especially in children, as being undoubtedly at times induced in the same way. In view of the numerous acute infections of the central nervous system, which had been pointed out by Dr Turner to be due to naso-pharyngeal infection of an acute nature, it was, in the speaker's opinion, possible that not a few chronic diseases of the nervous system were the result of a chronic infection from the nose or naso-pharynx. The former source has been pointed out by Dr Ford Robertson to be a frequent channel of infection.

Dr James Miller thought that examination by section in order to detect the presence of tuberculous changes was much more reliable than by inoculation. One could not help thinking that in a certain proportion of cases examined, the organisms may have been there merely temporarily, because people must be swallowing tubercle bacilli constantly. There was no doubt whatever that infection of the lung from cervical tuberculosis had been proved in a considerable number of cases in the way that Dr Philip had stated. The fact that there is pleurisy with adhesions between the parietal and visceral pleuræ at the apex is certainly in favour of that view; but, at the same time, there are other strong points against the transference of tuberculosis from the cervical glands to the lung. One view is that it is the bovine organism which is found in the glands of children, and is never found in the lung; or the organism is transferred from the bovine to the human type in the course of its passage to the lung.

Mr Dowden said it was a matter of no surprise that such a large area as the floor of the mouth should be a source of intoxication. It was only natural that infection should pass by the lymphatics into the lungs. 90-95 per cent. of children between 8 and 9 years of age have enlarged glands, and he would ask some of the path-

ologists who were in the habit of examining such cases, what proportion of tuberculous glands is found. The flat form of tonsil was less likely to be affected with tubercle ; it was the submerged tonsil which was associated with a great deal of infection. It was only natural that infection of the intestinal tract should be carried through the lymphatics. With regard to acute rheumatism he mentioned the case of a girl who had had a very bad sore throat, followed by definite symptoms of appendicitis, then the occurrence of acute rheumatism affecting various joints. Acute scarlet fever and conditions such as that are just what one would expect from a severe degree of sepsis of the mouth. He quite agreed with Dr Chalmers Watson that points of distant foci were sometimes missed.

Dr George Wilson (New Zealand), said he came here to-night to learn and had learned something, but he would like to ask Dr Logan Turner whether he had ever come across a new infection through the nose which has been a good deal discussed in the Pacific Islands ; he referred to the condition which is seen among the Maoris of New Zealand and amongst the Samoans of Samoa. The best way to explain the nature of this condition would be to give the history of a case. A Maori was sent to him from a district which was inhabited entirely by Maoris ; he came in order to be admitted into the private hospital, suffering, according to the diagnosis of the medical practitioner who was attending him, from rheumatic fever. He had an enlarged left knee and right elbow, with the ordinary infective temperature. The first striking point was that he appeared to be suffering from trichoma, and that was what suggested the real condition. The condition is a chronic infection of the nose, caused by a diplococcus, apparently a gonococcus, which is found in the mucous membrane of the nose and which infects the upper nasal passages and channels and gets into the sac ; it infects the sac and spreads up into the eye, producing a condition which is similar to trichoma. He (Dr Wilson) had been discussing the question with a German medical man a few days before, and this put him on the scent. A specimen was sent up to the pathologist, who found that it was a gonococcal infection. He would like to know if anything like that condition had been noticed anywhere else ? It is supposed to be produced by the Maori habit of rubbing noses—the Maori greets his friend by putting his nose against his friend's and keeping it there for some little time. One can imagine if one has a bad cold or a bad discharge how easily he may infect the other. He did not

suppose the matter would be of much interest to the medical men here, but a few years ago some Maoris went over to America, in order to demonstrate the Maori dance, but the whole of the little party were stopped at San Francisco because they were said to be suffering from trichoma ; it turned out to be the condition he had referred to.

Dr Webster thought there was no reason why infection should not be carried by the lymphatics. The reason why enlarged glands were so much more frequent in children than in adults seemed to be the greater changes that go on in the tissues of the young child. Tuberculous infections used to be more frequently found than they are now. Recently it had been shown that many conditions were the result of infection from the throat. Some of these cases he had mistaken for mumps ; in other cases it was soon apparent that the condition was tuberculous glands. Rheumatoid arthritis seemed to be due to infection from the mouth. The use of antiseptics would, he thought, greatly influence these conditions.

Dr Church said he had been using intravenous injections of iodoform in phthisis. He was greatly interested in what Mr Cathcart said about the transmission of infection to the lung from the joints. He (Dr Church) had a little experience from sanatorium practice in Edinburgh, and was greatly impressed with the intravenous injection of iodoform. In one case the temperature fell remarkably under the iodoform ; the sputum also decreased, and the general health of the patient is better. It seemed to him that Mr Cathcart's line of argument, that the absence of all glandular swelling and tuberculous manifestations, which are so common in phthisis, may be due to the antiseptic which is introduced, is the right one. Be that as it may, the case he (Dr Church) had under consideration showed improvement, and he hoped there would be still more improvement, by this method of treatment.

Dr Mitchell stated that he had recently been conducting a large number of observations on tonsils removed from cases of definite tuberculous adenitis. In these tonsils there were no naked-eye features suggestive of tuberculosis ; the lesion is invariably missed. On examination, however, giant cells are present and well formed, the caseation may be moderate, and the number of tubercle bacilli varies greatly. Some tonsils are flat, others are the small protruding

variety. In cases where tubercle is not suspected, microscopical evidence of tuberculous change is seen. The infection may pass along and produce lymphatic tuberculosis; it usually affects the basilar glands. There is no reason why the infection should not pass along for some distance before it produces a well marked lesion. He had examined something like 400 animals; in the guinea-pig, which is so susceptible, it is quite easily proved that the infection may pass a long distance before producing a well marked lesion. His statistics were not yet completed and he was not prepared to state the different types of bacillus that are found. The recognition of the part played by the bovine virus in tuberculous glands constituted a strong plea for the control of tuberculosis in cattle.

Dr Eason referred to one possible route of infection from the throat to the central nervous system which had not been mentioned. Dr Philip said that one must go back to the child, and Dr Shennan had referred to a route in the child which was not present in the adult. It is well known that the sphenoid develops from two centres. The complete union of these two foci does not take place till late in the child's life, and it may be that throughout life there is a vascular channel from the sphenoid. Sitelli has shown that there is a vascular communication through the sphenoid from the pharyngeal tonsil. There is perhaps a proportion of 6 per cent. of all cases where the pharyngeal duct remains patent. This condition is probably to be found more frequently in cases of acromegaly, but in quite normal people also the pharyngeal duct is found to be patent. In relation to the anatomical question, it is important to note that tuberculosis of the pituitary is a condition which has been described as primary; in all about 15 cases of tuberculous disease of the pituitary have been described. He had not had time to examine the literature, but there undoubtedly seemed to be primary cases of tuberculosis of the pituitary, and it seemed possible that the infection might be passed through in this way.

Dr Burn Murdoch was interested in the question of rheumatism. There was no doubt that many attacks of rheumatism are associated with infection from the throat. He had tried to investigate that point. It was quite remarkable how often one found this, and he mentioned the point in order to get others to give their experience. One gets cases of acute rheumatism with a subacute temperature;

it seems to be an obscure condition and one feels perfectly helpless. They don't respond to salicylates ; they seemed to wear themselves out—how, he did not know.

As regards tuberculous glands, his general impression. gathered from the Sick Children's Hospital, was strongly against glandular affections in the neck passing into the lung. He had not found lung infections common along with glandular infections of the neck. As regards treatment he strongly believed in getting diseased glands taken out, but he was quite confident that an enormous number of tuberculous glands in children could be recovered from without any surgical interference.

The *President* said the hour was late and he would not say much. There were many points connected with cerebral abscess, nose infection and so on which might be referred to. He entirely agreed with what Dr Burn Murdoch said with regard to acute rheumatism. For years past he had been firmly impressed with the fact that in many cases of rheumatism the infection arises from the throat, not so much sore throat at the beginning of the attack of rheumatism, but a history of sore throat some weeks before.

The question of infection from the cervical glands to the lung was exceedingly interesting. He never could see his way to accept Dr Philip's view on that point. The fact that cervical tubercle is so common in children, and phthisis so rare, seemed of itself to be quite conclusive, unless one grants, as had been suggested by Dr Miller, that the infective condition in the cervical glands is a bovine one, and does not affect the lung.

Suppurative tubercles in the neck go through the ordinary stages of tubercle, and if these glands are going to infect the lung this ought to occur in the child as well as in the adult.

Dr Logan Turner said it was too late to take up all the points raised, and to do so properly one would require a few minutes to plan out one's notes. He thanked the Society for the way they had received his paper ; he thought the secretaries were justified by the discussion in having selected this subject. One would think from all that had been put forward that the upper air passages were the source of all the ills that flesh is heir to. It suggested that an ear and throat specialist should be attached to every institution, or, at any rate, that all physicians and surgeons should have a very special knowledge of the nose and throat.

As regards the discussion as a whole, tubercle has formed the most important point of interest, as was to be expected. There is no doubt that the lymphatics or the lymphatic ring is a source of infection, but whether that infection spreads throughout the lymphatic glands of the neck and infects the lung or not is a point open to greater discussion. The evidence is rather against that being so; personally he would be against it. He thought Dr Philip's picture a very pretty one and easy to work out, but he did not think there was sufficient definite proof of such infection. One would expect to get evidence of direct continuity between the glands and lung apex at post-mortems and operations. He quite agreed with those who regard the small tonsil as the dangerous one; there is no doubt that in cases where there is tubercle of the tonsil and cervical glands that is the form of tonsil which is met with.

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